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ATROPHY OF THE OPTIC NERVE AND NAEVUS FLAMMEUS ASSOCIATED WITH HEMANGIOMA OF THE CHOROID

REPORT OF A CASE

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The following case is reported because of the possible relation of the condition to encephalotrigeminal angiomatosis, which was recently reviewed by Appelmans.[1]

REPORT OF CASE

History.—A. C., a boy aged 12 years, came to the Birmingham and Midland Eye Hospital on Dec. 30, 1935, complaining of pain in the left eye of three weeks' duration. The left eye had been blind since birth, and on occasions it had been painful for short periods.

Examination—The condition noted on examination was as follows:

Right Eye: Vision was 6/6. The eye was structurally and functionally normal. There was no evidence of abnormal vessels. The tension was 17 mm. of mercury (Schiötz). The field of vision was full as determined by perimetry with the use of a 0.5 degree red test object and a 0.5 and a 0.25 degree white test object at 0.3 meter.

Left Eye: Vision was reduced so that there was no perception of light. There was a deep zone of dilated ciliary blood vessels forming a prominent ciliary flush. In addition, the conjunctival vessels were dilated. The cornea was diffusely edematous: the anterior chamber was shallow and in its lower part presented a small hyphema. The vessels of the iris were prominent, and several large vessels were observed coursing irregularly on the anterior surface. The lens was intumescent and opaque. The tension was 42 mm. of mercury (Schiötz). Transillumination gave an even reflex, but this was somewhat more diminished than the opacity of the lens would lead one to expect.

Face: The face presented the condition of naevus flammeus limited strictly to the left side of the midline; the extent of the distribution is best seen in figure 1; briefly, it involved chiefly the distribution of the maxillary division of the trigeminal nerve, with some overlapping in the area of the ophthalmic division. The mucous membranes of the left cheek, the left side of the upper jaw and the hard and soft palate were involved.

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1. Appelmans, M.: Encephalotrigeminal Angiomatosis, Arch. d'opht. **52**:835 (Dec.) 1935.

Body: The body showed numerous areas of hemangiomatosis. A complete neurologic examination was carried out, but no lesions involving the nervous system were discovered.

Roentgenograms of the Skull: There was no visible area of calcification in any part of the skull. The left optic foramen was slightly larger than the right.

Enucleation.—On the basis of the history of the recent incidence of the attack of pain in the left eye present at the time of admission, and on the basis of the result of examination on transillumination, it was felt that in all probability the present state of the eye was due to hemorrhage from a choroidal hemangioma. Excision of the eye was advised and was performed, without unusual bleeding, on December 31, with the patient under general anesthesia. Recovery was uncomplicated, and healing of the socket was normal.

Pathologic Examination.—The enucleated eye was subjected to pathologic examination. The report from the department of pathology is as follows: "Sec-

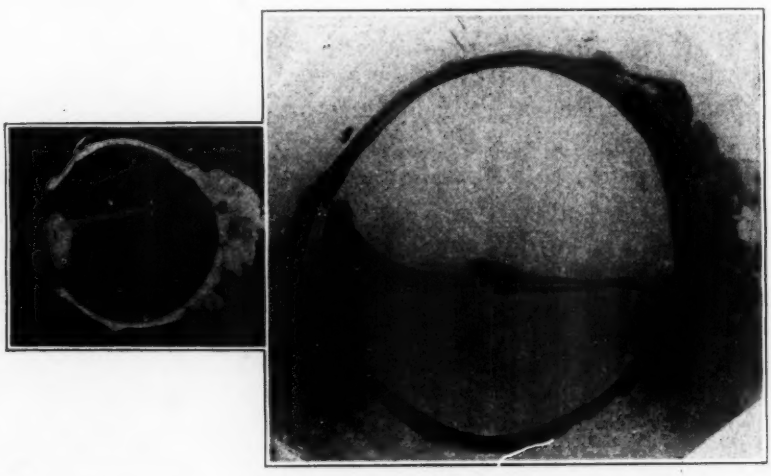

Fig. 2.—Hemisections of the left eye, showing the opaque lens pushed forward, the retina folded up into a central cord of tissue and completely detached and the flat hemangioma of the choroid occupying the posterior part of the globe.

tion shows keratitis with vascularization of the cornea; anterior synechiae; degenerated lens; detached retina; large angioma of choroid, and cupped disk" (fig. 2).

The microscopic study of the sections showed a number of points:

1. The filtration angle of the anterior chamber was completely closed by the forward projection of the lens and iris (fig. 3).

2. The optic disk was deeply cupped, and the retina was seen to be attached to its margin (fig. 4).

3. The optic nerve was diffusely infiltrated by small hemorrhages (fig. 5).

4. The retina, which was greatly degenerated, had been completely detached, except at the margin of the disk, and was seen as a double fold passing forward in the center of the globe. The space around was, in the fresh state, occupied by subretinal fluid and hemorrhage.

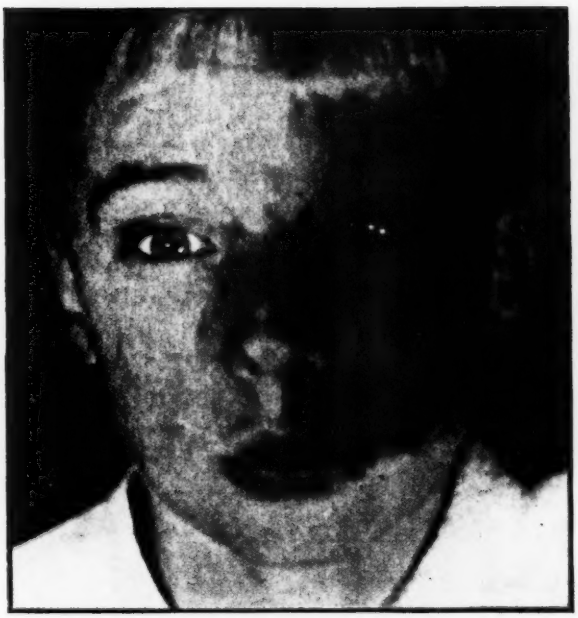

Fig. 1.—Colored photograph of the patient.

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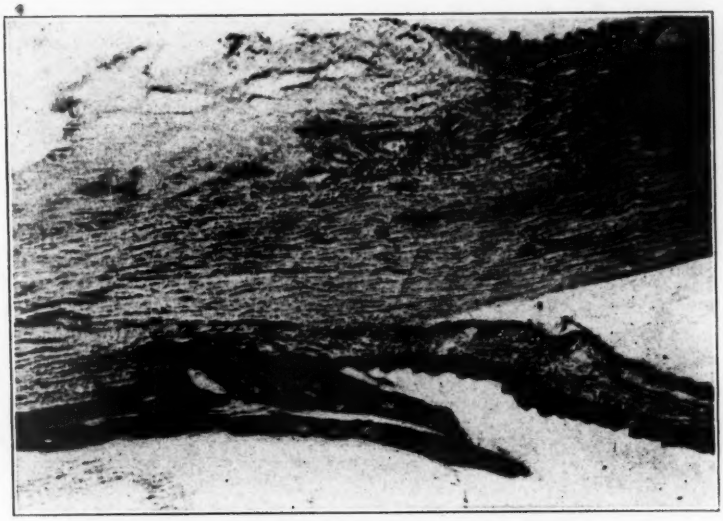

Fig. 3.—Photomicrograph of a section of the filtration angle.

Fig. 4.—Photomicrograph of a section of the optic disk.

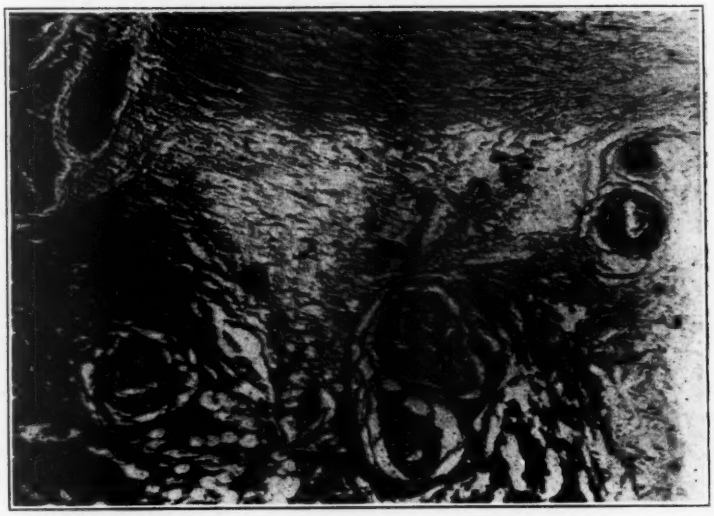

Fig. 5.—Photomicrograph showing small hemorrhages in the optic nerve.

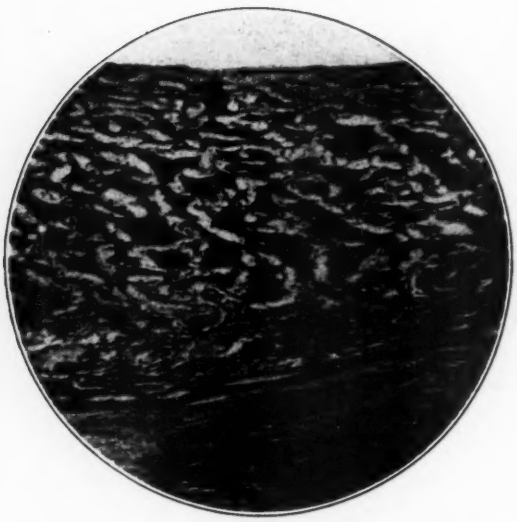

Fig. 6.—Photomicrograph of a section of the hemangioma of the choroid.

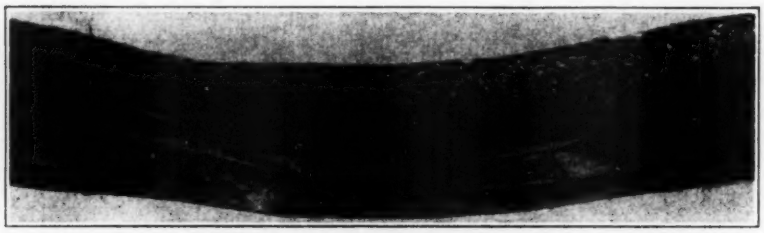

Fig. 7.—Photomicrograph showing the path of the neoplastic tissue.

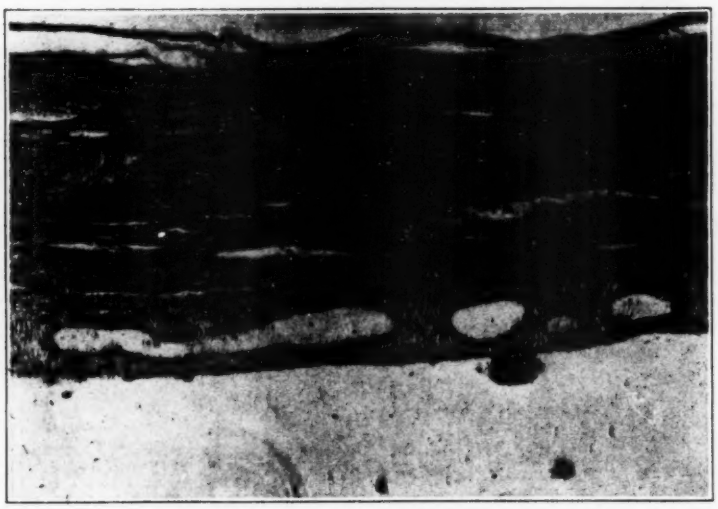

Fig. 8.—Photomicrograph of a section of episcleral tissue, showing the presence of dilated vessels.

5. In one hemisection of the globe a circular, ill defined mass was seen projecting slightly from the surface of the choroid; its color was reddish brown. Portions of this tissue, on section, proved to consist solely of blood clot.

6. The posterior half of the choroid was seen to be greatly thickened, the widest part corresponding with the posterior pole; on section this thickening was seen to be composed of a diffuse hemangioma (fig. 6).

7. The lens was cataractous and swollen, but the changes were of recent origin.

8. Serial sections of the globe showed the path of entry of the neoplastic tissue. This followed the course of one of the venae vorticosae. Its path from the growth of the choroid, in the region just behind the equator of the globe, could be seen passing obliquely backward through the sclera and thence outside the globe to one of the greatly dilated vessels lying to the side of the optic nerve (fig. 7).

9. The episcleral tissue also showed the presence of many very dilated vessels which appeared to have the structure of veins (fig. 8).

COMMENT

From the history of attacks of pain in this case it seems probable that hemorrhages from the choroidal hemangioma, with rise of tension, had occurred previously from time to time. The advanced condition of degeneration of the retina and the completeness of its detachment lend color to this supposition. Consequently, one is not in a position to confirm the findings of Thiel that cupping of the optic disk and atrophy of the optic nerve are independent of tension. On the other hand, the fact that the patient went for long periods without pain in the eye and that the intumescence of the lens was of recent development suggest that, apart from the few occasions mentioned, the tension in the eye was not raised, and it is extremely probable that so advanced a degree of cupping is the result of essential atrophy of the optic nerve, consequent on defective vascular nutrition, and is not the result of prolonged increase of the intra-ocular pressure.

THE JAW-WINKING PHENOMENON

REPORT OF A CASE

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It is with some hesitation that I report a case of the jaw-winking phenomenon, since I know full well that it has little clinical value and that the condition is so rare as to be possibly unimportant. However, it is felt that the rarity and curiousness of the disorder are in themselves sufficient reason for bringing it to the attention of ophthalmologists.

The condition which has been called, rather inelegantly, the jaw-winking phenomenon was first described by Marcus Gunn in 1883, when he showed a patient with this disorder before the Ophthalmological Society of the United Kingdom. Since that time about ninety additional cases have been reported, although there is some reason to question the authenticity of some of them. In 1895 Sinclair [1] collected a number of instances and discussed several possibilities as to the etiology. Sym [2] in 1908 reported a case and again reviewed the etiologic considerations. The review which I made of the literature does not pretend to be complete, but it is hoped that a summary of a few of the more typical cases, together with a description of the present case, will serve to affirm the opinion that the phenomenon is a definite clinical entity.

REPORT OF CASE

A woman of 41 years presented herself, complaining of difficulty with near work and considerable lacrimation. She showed a moderate amount of compound hyperopic astigmatism in each eye and slight presbyopia, for which glasses were prescribed. The rest of the examination gave entirely negative results except for a very small amount of ptosis of the upper lid of the right eye. This was manifest only when the eyes assumed a position of rest, looking straight ahead (fig. 1 *A*). When the eyes looked upward the eyelid readily retracted, showing no paralysis. The ocular movements were unlimited in all directions. There was no evidence of Horner's syndrome to which the ptosis could be attributed. With a red glass no diplopia could be elicited. The pupils reacted directly and consensually to

1. Sinclair, Walter W.: Abnormal Associated Movement of the Eyelids, Ophth. Rev. **14**:307, 1895.

2. Sym, William George: A Case of Partial Ptosis with Exaggerated Involuntary Movement of the Affected Eyelid: The "Jaw-Winking" Phenomenon, Ophth. Rev. **27**:197, 1908.

light and normally in accommodation. The patient stated that the ptosis had existed for as long as she could remember. She stated also that whenever she ate or chewed gum vigorously the upper lid of the affected eye "winked," that is, the upper lid moved up and down rapidly. This curious retraction of the upper lid was evidenced when she merely moved the lower jaw downward and was much more marked when she moved the lower jaw to the left (fig. 1 *B*). Movement of the jaw caused no winking or jerking of the left eyelid. The condition caused the patient little concern, since it was apparent only when she executed rather vigorous movements of the lower jaw. There was no history of a similar condition in her immediate family, but she had been told when she was a little girl that she had an eye "just like" her aunt's.

Fig. 1 (the author's case).—*A*, the aspect at rest; *B*, the aspect on movement of the jaw to the left.

Fig. 2 (case of Sym [1908]).—*A*, the aspect at rest; *B*, the aspect during sudden depression of the jaw.

CASES REPORTED IN THE LITERATURE

In 1908 Sym reported the case of a woman of 30 years. The only fault which he found with the eyes was that which is well illustrated by figure 2. There was a distinct degree of ptosis of the left upper eyelid (fig. 2 *A*). The levator muscle was not paralyzed, as was shown by the fact that the patient could elevate the lid readily on looking upward. Movements of the globe were perfect. When, however, the patient was chewing her food or when she was singing, the upper lid might almost be said to spring upward. When she suddenly opened her mouth by depressing the lower jaw, the lid sprang up in the manner shown in figure 2 *B*. When the lid flew up there were no

corresponding movement of the right upper lid, no corrugation of the forehead and no untoward movement of the eye itself. There was no evidence of Horner's syndrome.

In 1912 Thomson and Souter [3] reported a case of this condition in a girl of 9 years (fig. 3 *A*). When the eyes were at rest and looking forward the patient showed a slight degree of ptosis of the left upper eyelid. She showed no miosis and no evidence of unilateral sweating or flushing. The ocular movements were normal in all directions. When the mouth was opened slowly there was a very faint upward jerk of the left upper lid, but when the mouth was opened rapidly and worked to the right there was a marked upward jerk of the lid (fig. 3 *B*) and 2.2 mm. of the sclera was exposed above. When the jaw was worked to the left there was no movement of either upper lid. The patient was unable, while looking forward in an ordinary way, to raise either eyelid without using the frontalis muscle or opening her mouth, but the lids became elevated equally with upward move-

Fig. 3 (case of Thomson and Souter [1912]).—*A*, the aspect at rest; *B*, the aspect on movement of the jaw to the right.

ments of the eyes. The lids closed equally well, both in sleep and on forced closure. General physical examination, both of this patient and of the one previously mentioned, revealed nothing significant.

Wilkinson [4] and Coburn [5] each reported a case of similar elevation of an upper lid associated with movement of the lower jaw. Coburn's patient was unable to open his left eye, the one affected, except when he opened his mouth. Wilkinson's patient was able to open and close the eyes normally, except that when he made an extra effort the right, or affected eye, opened a little wider. In a case reported by Lutz [6] in

3. Thomson, J. E. G., and Souter, W. C.: Congenital Ptosis with Associated Lid Movements of the Affected Eye, Ophth. Rev. **31**:172, 1912.

4. Wilkinson, cited by Wood, Casey A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1913.

5. Coburn, cited by Wood, Casey A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1913.

6. Lutz, Anton: Jaw-Winking Phenomenon and Its Explanation, Arch. Ophth. **48**:144 (March) 1919.

1919 the phenomenon in question was first noticed at the age of 5 years. There was a slight ptosis on the right side, and with a colored glass diplopia could be elicited, corresponding to that caused by paresis of the right superior oblique muscle. Elevation of the right upper eyelid could be induced by opening the mouth or by moving the jaw from side to side, but the patient could also elicit the movement voluntarily, without any movement of the jaw. The latter finding is a peculiarity which was not present in any of the other reported cases. It is impossible normally to elevate one lid alone.

In Holloway's[7] case of a woman aged 44 there was a history of distinct drooping and retraction of the right lid at times, or, as the patient expressed it, she winked while she ate. The fields showed diplopia and indicated paresis of the right superior rectus muscle. The eyes were otherwise normal. Two additional cases were reported, one by Kleinhans and one by Menacho. In both these patients, who had ptosis of one eyelid, the lid shot upward when the mouth was opened, but lateral movements of the jaw had no such effect.

In the reported cases the error was about equally distributed between the right and the left side. No case was reported in which both sides were involved. Males are affected about as often as females. In most cases ptosis was present on the affected side, although a few cases were reported in which no ptosis was evident.

Several explanations have been offered as to the probable meaning of these associated movements. After Gunn made the first report of a case of this condition a committee was appointed by the Ophthalmological Society of the United Kingdom to consider it. The explanation offered by this committee is by far the most satisfactory submitted, namely, that the levator muscle receives motor impulses from both the third and fifth nerve. Sym concluded his paper on the subject as follows:

> There does not appear to be any other probable explanation of this curious association than that in some inexplicable way there arises some confusion in the joining up of fibers and cells belonging to the fifth and third nuclei in such a fashion that the levator receives less than its normal innervation, and there is, therefore, a certain degree of ptosis, but without any paralysis of the muscle, which is capable of full contraction. At the same time the levator receives some fibres which are "intended for" one or more of the muscles of mastication and when these muscles are put in action, at all events when put strongly in action, the levator is unintentionally innervated, producing the curious effect described.

This explanation is anatomically conceivable, since the nuclei of the third and the fifth nerve closely approximate each other as they lie

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7. Holloway, cited by Wood, Casey A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1913.

in the floor of the sylvian aqueduct, and they are connected by the posterior longitudinal fasciculus. That the lesion is either nuclear or very close to the nuclei is shown by several cases in which the patient showed paresis of one or more ocular muscles. Lutz agreed that the lesion was probably supranuclear and very close to the nuclei but expressed the belief that it had to do with the inhibitive mechanism of the levator. He has published an excellent review of the various theories. Menacho observed that in his case and in one or two other reported cases, the condition appeared to be acquired rather than congenital, and he stated the opinion that an exclusively anatomic explanation was unjustified. He recognized the close relationship which exists between the functions of the cranial nerves and argued that the condition was due to an alteration of this functional relationship rather than to an anatomic alteration. When a review of the published cases is made, however, it is noticed that in the majority the condition is described as congenital and in the remaining few it is said that the condition "was not noticed" until some time after birth. The fact that the condition was not noticed does not disprove its existence.

Bishop Harman attempted to show that the condition is an atavistic anomaly. He thought it was merely a manifestation of a reversion to a primitive type, such as, specifically, the shark, in which the cranial nerves are more closely associated than in man. His conclusions are not, it must be confessed, very convincing.

Ole Bull considered that such a movement is purely a reflex and what is constantly seen in children with blepharospasm who open their mouth when they wish to open their eyes. This theory can be objected to on the ground that in all cases only one eye is affected and that in some cases the associated movement of the lid is elicited only on lateral movement of the jaw.

There have been cases reported under the name "jaw-winking" which are not true instances of the condition. Schirmer reported the case of a man aged 28 in whom facial paralysis on the left had developed. Four months later he began to show slow improvement. After three more months he was unable to wink his left eye, although the nerve supply to the other facial muscles showed considerable evidence of recovery. It was noted, however, that movements of the left cheek, in laughing or chewing, were accompanied by partial closure of the left eye. The reporter felt that these associated movements were due to the fact that fibers from the central stump of the facial nerve that were intended for the cheek muscles had grown into the orbicularis muscle. There was no involvement in this instance of the muscles used in mastication or of the levator palpebrae superioris, so the case cannot be considered a true case of jaw winking.

A similar case was reported in January 1937 by Temple Fay [8] The patient was a woman aged 42 who had had a transplantation of the facial nerve on the right. She had previously had facial paralysis on the right after operation on the mastoid. After sixteen months the facial nerve on the right showed some evidence of regeneration but with a rather bizarre result. Dr. Fay stated:

> When the command to close the right eye was given, not only did the right corner of the mouth draw up, but even with the wink reflex on the left, there was a definite jerk or twitching at the lower angle of the mouth on the right. . . . When the patient forcefully closed the eye, the effort gave a response in the lower ⅔ of the face. When instructed to draw up the corner of the mouth, the patient showed a slight twitching movement beneath the right eye.

In this case, as in the one previously cited, neither the muscles used in mastication nor the levator muscle was involved, and the case cannot be considered a true case of jaw winking.

In none of the articles reviewed was any mention made of treatment, and in many of the cases, including the one reported by me, treatment was neither desired nor advisable. It might not be out of place to suggest, however, that in those cases in which ptosis is pronounced and in which there is no paresis of the superior rectus muscle a tenotomy of the levator muscle might be done, to be followed later by transplantation of one third of the tendon of the superior rectus muscle to the upper lid (Motais' operation). Such a procedure would produce a satisfactory cosmetic result and at the same time provide for functional action of the upper lid.

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8. Fay, Temple: Jaw-Winking, Arch. Neurol. & Psychiat. **37**:208 (Jan.) 1937.

LOCALIZATION OF INTRA-ORBITAL FOREIGN BODIES

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PRINCIPLES OF ROENTGEN TECHNIC

The localization of intra-ocular foreign bodies is a rather special department within the general field of roentgenology and involves many problems which are of great importance to both the ophthalmologist and the roentgenologist. The ophthalmologist is far too prone to accept without question the indicated position of a foreign body on a chart showing the localization which has been handed to him from the roentgenologic department. This chart represents as accurately as possible within the scope of the method used and the experience of the roentgenologist the position of the foreign body in relation to the ocular structures. There are, however, so many possible errors that it is to be highly recommended that the ophthalmologist thoroughly familiarize himself with the principles involved and the methods used, so that he may follow the procedure intelligently and not accept the data on the localization chart at face value.

There is no problem in medicine requiring mutual cooperation to a greater degree than that of the localization of an intra-orbital foreign body. It would be a matter of relative simplicity were the globe visualized on the x-ray film, but such is not the case, and as there are many bony structures superimposed over the area of the globe the problem becomes complex.

It is important for the ophthalmologist to have definite data on the following points in regard to an intra-orbital foreign body: (*a*) whether it is visible on roentgen examination; (*b*) its approximate size; (*c*) its relation to the structures of the globe; (*d*) whether it is intra-ocular or extra-ocular.

The fact that numerous methods and apparatus are used for localization, some workers relying on one method and others on a different set of principles, shows clearly that no single procedure can be relied

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From the X-Ray Laboratory of the Wills Hospital.

This paper is an abridgment of a thesis submitted to the faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Doctor of Medical Science for graduate work in radiology.

on for accuracy in all cases. The roentgenologist is often too prone to accept whatever practice he has been taught in his period of training or is prevalent in his community. The ophthalmologist is also likely to accept without question the results obtained on a diagrammatic chart. My associates and I do not recommend adherence to any single procedure of localization exclusively but believe that if both these specialists understand and consult together regarding the problems presented by their cases the accuracy of the results will be greatly improved.

The underlying principles of the roentgen rays as applied to this special problem are similar to the general principles of roentgenography, but assume a far greater importance from a geometric standpoint because of the extreme accuracy required. I shall therefore briefly point out the methods and factors entering into this problem which I believe would assist the ophthalmologist in his interpretation of the result.

ROENTGENOGRAPHIC METHODS

1. *Simple Method.*—This consists of two roentgen projections at right angles to each other, from the postero-anterior and the lateral aspect. This merely identifies the presence of the foreign body and shows its general relationship with the bony structures of the orbit. At the present day this is recommended only as a means of identification. Such a method of localization was the first reported in the literature, having been described by Clark [1] in 1896.

2. *Geometric Methods.*—These localize the position of the foreign body in relation to some fixed marker outside of the globe, which is placed in a known relation to the plane of the cornea. These methods are the most commonly accepted, and most of them are sufficiently accurate for ordinary work. The first of these methods to be reported was described by Sweet [2] in 1898.

3. *Physiologic Methods.*—These depend on the rotation of the globe between predetermined points of visual fixation and the resulting behavior of the foreign body with regard to the optic center of the eye. An example of this type of method was reported by L. Webster Fox [3] in 1902 and another by Köhler [4] in 1903.

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1. Clark, C. F.: A Question as to the Presence and Location of Minute Fragment of Steel in Eye Determined by the Roentgen Rays, Tr. Am. Ophth. Soc. **7**:711, 1894-1896.

2. Sweet, W. M.: The Roentgen-Rays in Ophthalmic Surgery, Am. J. M. Sc. **116**:190, 1898.

3. Fox, L. W.: The Localization of Foreign Bodies in the Eye, Philadelphia M. J., 1902; Lancet, 1902.

4. Köhler, A.: Zur Technik des Fremdkörpernachweises im Augapfel, Fortschr. a. d. Geb. d. Röntgenstrahlen **6**:190, 1903.

4. *Roentgenographic Stereoscopic Methods.*—Several procedures have been recommended, one of which was reported by Béclère and Morax [5] in 1907. We found these to be less accurate than the other standard methods because of the narrow angulations involved and because they require considerably more experience for the same degree of accuracy to be attained.

5. *Accessory Methods.*—These include: visualization of the anterior portion of the globe free from interference by the bony structures; the injection of air or opaque material into the capsule of Tenon to identify the posterior part of the sclera; autovisualization, in which the patient identifies and checks the location of the foreign body by the sensitivity of his retina to the roentgen rays, and the methods in which various markers are used which have been previously introduced in the conjunctival sac or sewn to the lids to assist in identifying the plane of the cornea.

Comment.—All the principles and methods just described should be thoroughly known by the radiologist who attempts to assume responsibility in reporting on cases of an intra-ocular foreign body. This does not mean that he must be familiar with every device, but if one method leaves doubt in the mind of the ophthalmologist the roentgenologist should be able to clear this up by using the principles of whatever other procedure is required to settle the problem at hand.

The underlying geometric principles as applied to any of the methods already described are of supreme importance, and failure to observe these means inaccurate work. Therefore, the ophthalmologist will find an understanding and application of the following simple geometric principles of the greatest value in checking the roentgenographic procedure.

Principle 1: A false impression is given as to the size of a foreign body owing to magnified distortion (fig. 1 *A*). Because of the spreading of the roentgen rays, which obey the law that the intensity of light is inversely proportional to the square of its distance from the source, the resulting image is magnified.

Principle 2: A false impression is given as to the size of the foreign body owing to true distortion, if its axis is not parallel to the plane of the x-ray film (fig. 1 *B* and *C*). The actual working conditions with a modification of the localizer of Sweet are represented in figure 1 *D*, from which one can readily see that the measurement and the size of the foreign body on the x-ray film may be misleading.

5. Béclère and Morax: Un nouveau procédé de localisation des corps étrangers métalliques intra-oculares par la stéreo-radiographie avec repères cornéens, Rev. gén. d'opht., 1907.

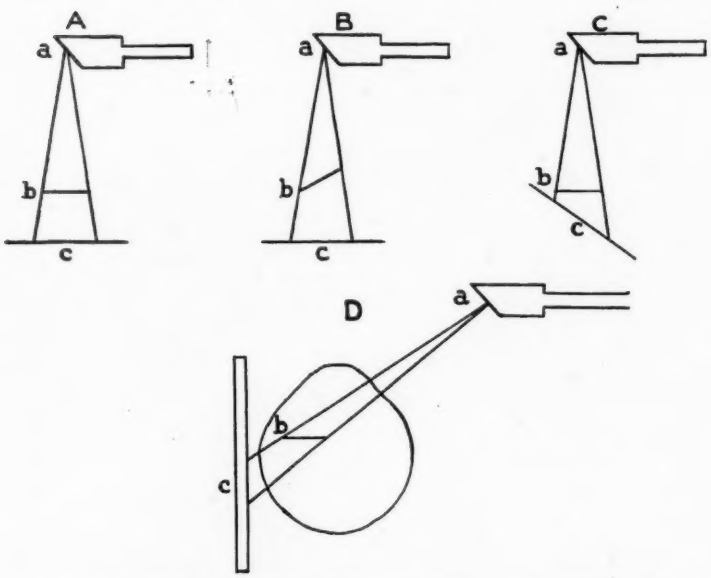

Fig. 1.—*A*, magnified distortion due to spreading of the beam of the roentgen rays. *B*, true distortion due to the angle of the foreign body. *C*, true distortion due to the inclination of the surface of the x-ray film. *D*, relationship of the x-ray tube, the eyeball and the film in the modified method of localization of Sweet. The letters have the following significance: *a*, the target of the x-ray tube; *b*, the foreign body, and *c*, the x-ray film.

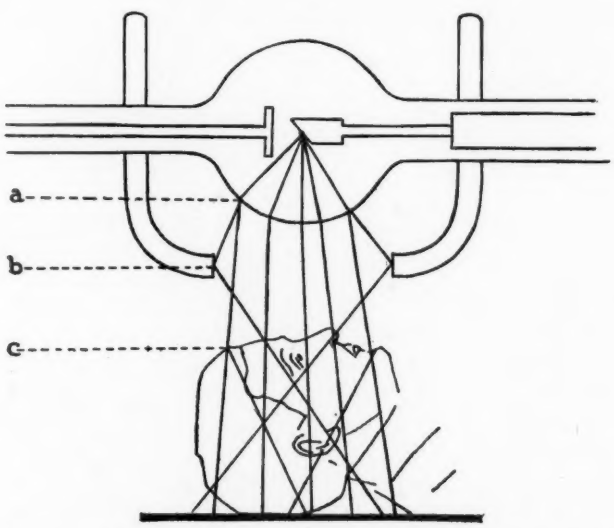

Fig. 2.—Diagram showing scattering of the beam of the roentgen rays wherever it strikes against a hard surface. The main points from which this scattering occurs are: the glass bulb of the x-ray tube (*a*), the lead glass or metallic container (*b*), and the tissues of the patient's head (*c*).

These factors of distortion are minimized by putting the greatest practicable distance between the x-ray tube and the surface of the film, which makes for more nearly parallel beams of radiation, and also by the approximation of the injured eye as near as possible to the surface of the film, which gives a sharper image on the resulting roentgenogram.

Axiom 1. In all cases of localization of a foreign body the apparatus should be so constructed as to have the greatest practicable distance between the tube and the film and the smallest possible distance between the injured eye and the film.

Principle 3: Definition or sharpness of outline on the roentgenogram depends on the minimum of distortion, the smallest possible degree of scattered radiation, perfect immobilization of both the head and the eye and a fine effective focal spot in the target of the x-ray tube.

Scattered rays tend to blur the outline. The scattered rays are generated from the x-ray tube, the walls of the holder and the head of the patient (fig. 2). This scattering is avoided by using the smallest possible opening for the emission of the roentgen rays.

If one were dealing with a point source from which the roentgen rays were emitted, one would have to deal only with distortion, scattering and movement, but as the focal spot of the roentgen rays is of a measurable diameter the foreign body is often smaller than the source of the rays. This brings an important factor into the problem. The resulting phenomenon is similar to that of the heavenly bodies. In an eclipse of the sun, in which the moon passes between the sun and the earth, the moon casts a shadow on the surface of the earth in which the central portion, or the zone of total eclipse, is denser than the periphery, or the zone of partial eclipse. The area of total eclipse is the umbra, the peripheral zone being the penumbra. A similar phenomenon is present in the localization of a small intra-ocular foreign body, because often the size of the foreign body is smaller than the diameter of the effective focal spot and the x-ray tube. This causes a dense central shadow, or umbra (fig. 3 *A*), and a peripheral zone of decreased density, or penumbra (fig. 3 *B*). As the penumbra is not visible through the bony structures it may be readily understood that many minute particles are rendered invisible on the x-ray film, irrespective of their density.

Axiom 2. In the localization of an intra-ocular foreign body the greatest care must be taken to secure perfect immobilization of the head and eye, use of the smallest aperture practicable for the emission of the beam of the roentgen rays and the finest possible effective focal spot within the x-ray tube to be used.

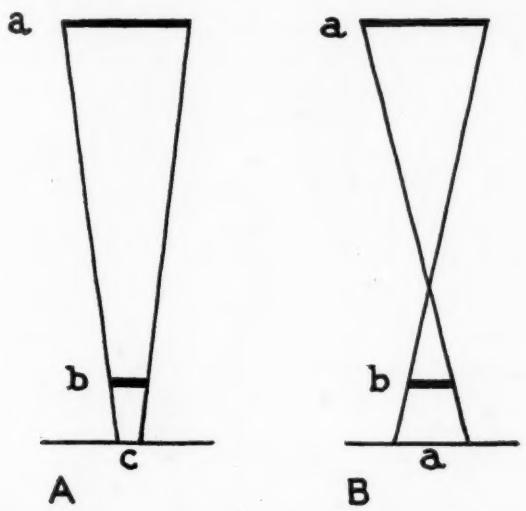

Fig. 3.—*A*, diagram illustrating the umbra phenomenon, in which *a*, indicates the focal spot of the x-ray tube; *b*, the foreign body, and *c*, the x-ray film. The foreign body is smaller than the diameter of the focal spot, or source of radiation. The resulting image on the surface of the film (*c*) is smaller than the foreign body (*b*). *B*, diagram illustrating the penumbra phenomenon, in which *a*, indicates the focal spot; *b*, the foreign body, and *c*, the size of the penumbra. The penumbra is larger than the size of the foreign body and causes a zone of relatively decreased density surrounding the actual image, or umbra.

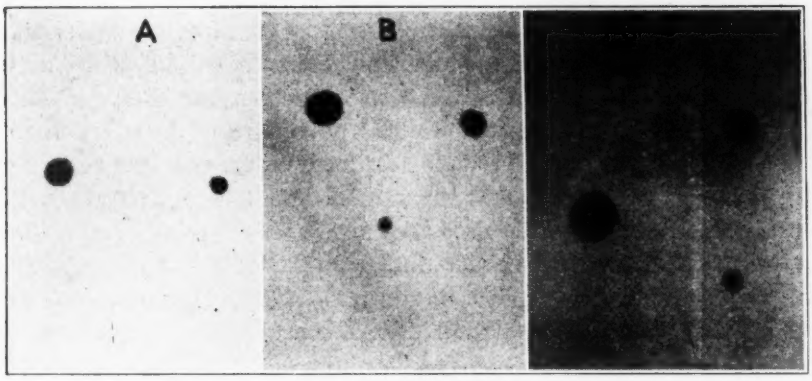

Fig. 4.—*A*, roentgen ray projection of three lead particles, with the x-ray tube at a distance used with the modified Sweet localizing apparatus. The focal spot of the tube is approximately 1.1 mm. in diameter. The distance of the foreign body from the surface of the film is approximately the same as in the actual localization practice on the patient. *B*, roentgen projection secured under conditions similar to those for *A*, except that a focal spot of 3.2 mm. was used. *C*, roentgen projection secured under conditions similar to those for *A*, except that a focal spot of 5.5 mm. was used. Notice the effect on the outline of the foreign body. As the focal spot increases, the outlines become indistinct.

GEOMETRIC LOCALIZATION

A discussion of the underlying principles used in the geometric methods of localization of an intra-orbital foreign body is essential to an intelligent understanding of the problems involved. I seek especially to bring out those factors which cause errors in accuracy so that the ophthalmologist may be on his guard in criticizing the results obtained by any of these methods. The most commonly understood and generally accepted geometric method is either the original method of Sweet or the more recently evolved modified Sweet procedure. The apparatus in either case is built as an instrument of precision and embodies in its use many factors with which the roentgenologist must be thoroughly familiar. This is understood in the larger clinics, where trained roentgenologists see abundant material, but in the small medical centers, where a case of an intra-orbital foreign body is met with only occasionally, it is more difficult for the radiologist to familiarize himself with the apparatus and method sufficiently to understand its strong and weak points. These I hope to bring out in the following discussion.

To define the position of any point in space with relation to another, three coordinates are required, corresponding to the three geometric planes in space. In considering the globe of the eye I refer to these three planes as the equatorial diameters and name them, according to anatomic terminology, the horizontal, the sagittal and the coronal plane. The intersection of these planes is the optic center of the globe, and a knowledge of the distance of the foreign body from this optic center in relation to the three spatial planes definitely localizes it in relation to the ocular structures. The modified Sweet method will be discussed to illustrate the principles of geometric localization, but these principles apply to all geometric methods. In this procedure of localization it is presupposed that the eyeball remains stationary during the examination, and a marking device is placed in a fixed and known relation to the optic center. In the modified Sweet apparatus this is done by accurately centering a small metallic marker directly in the line of the optic axis, which is the center point of the pupil, and exactly 10 mm. anterior to the tangential plane of the cornea. The mechanical construction of the instrument assures this constant relationship of the marker to the globe. It also embodies in its construction a constant distance between the target of the x-ray tube and the surface of the film, a constant inclination of the beam of the roentgen rays and a constant shift of the tube, or the formation of a parallax angle. With these factors known and predetermined, one is able to define the position of a foreign body in relation to the marker and calculate its relation to the optic center.

Two views are made. The first is taken with the target of the x-ray tube in exact alinement with the marker, which allows one to measure

accurately the distance of the foreign body posterior to the marker and thus determine its distance anterior or posterior to the coronal equatorial plane. The same view allows one to determine the distance above or below the horizontal equatorial plane which is also indicated by the position of the marker. Only one more measurement is required to identify the position of the foreign body within the globe, this measurement being the distance to the temporal or nasal side of the sagittal equatorial plane. To obtain this the x-ray tube is shifted parallel to the surface of the film a predetermined distance, and a second exposure is made. The parallax shift forms two series of triangles, in one of which series the marker is the apex of the triangles, the foreign body being the apex in the other series. The series in which the marker is used may be illustrated by figure 5, in which the distance of the marker from the surface of the film can be easily determined by the formula $X = \frac{BC}{A + B}$. The series in which the foreign body is used as the apex may be illustrated in a similar manner, the distance of the foreign body from the surface of the film being calculated by the same formula, as this formula applies to the height of similar triangles, irrespective of their inclination. It is therefore possible to determine accurately the position of the foreign body in relation to the temporal or nasal side of the sagittal equatorial plane. As these planes intercept at the optic center, one has the relation of the foreign body to the optic center, and the localization is complete.

It now remains to chart the findings so that there will be a permanent record. It will be noticed that the chart reconstructs the actual operating conditions and factors embodied in the apparatus for localization. Figure 6 shows the apparatus as viewed from the end and indicates the position of the marker, the inclination of the beam of the roentgen rays and the position of the film in relation to the eyeball. Measurements made from the x-ray film are indicated on the chart in column *E*. These measurements, however, are not in millimeters, because of the magnified distortion, but are made in arbitrary units which correspond to millimeters. The key plate used to measure these arbitrary units is a handy, convenient and accurate manner of transferring them from the film to the chart. Similar measurements are made, the columns indicated across the top of the key plate being used to transfer to corresponding columns on the localization chart. Lines are retraced parallel to those indicating the beam of the roentgen rays and where these cross the foreign body is located. Thus one has established a certain set of known and predetermined conditions in using the instrument for localization and has merely reversed these conditions in charting the results of the procedure. In all geometric methods this general type of procedure is followed, with various minor modifications.

There are several possible sources of error inherent in the apparatus, such as deviation in alinement of devices for centering, carelessness in perfect centering and alinement of the tube, slight movement of the

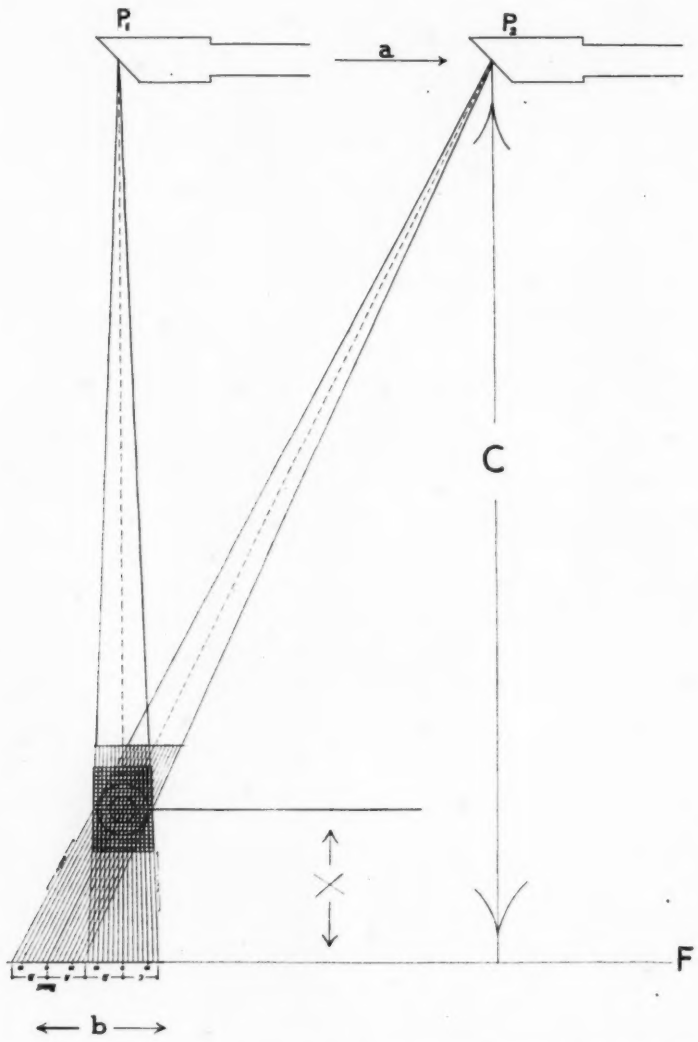

Fig. 5.—Relation of the localization chart to the x-ray tube as viewed from above. The tube is shifted from the point P_1 to the point P_2. *a* represents the direction of the shift; *b*, the parallax shift of the image on the x-ray film; *C*, the distance of the x-ray tube from the surface of the film (*F*), and *X*, the distance of the marker from the surface of the film.

apparatus while shifting the tube and inability of the patient to maintain ocular fixation, especially in cases of nervousness, photophobia or injury of the eye. The procedure must be carried out with meticulous

care and repeated if there is any question as to the findings. We always insist that two workers make independent measurements and charts, which must agree perfectly before the results are acceptable.

If the foreign body is small, a single central point is chosen, but if it is sufficiently large, two points, usually anterior and posterior, are localized, different colored inks being used.

In the procedure just described the foreign body has been localized, and its position has been indicated on the chart provided for this purpose (fig. 7), which represents the usual type of chart used in the

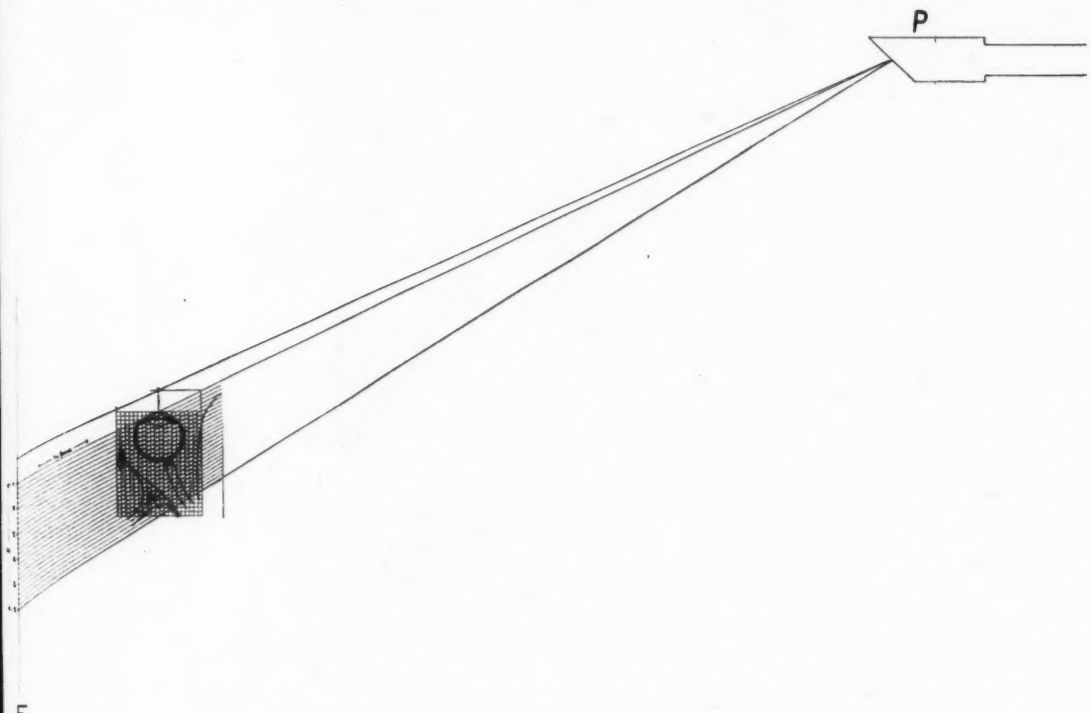

Fig. 6.—Relation of the localization chart to the x-ray tube as viewed from the side, indicating the angle of inclination of the beam of the roentgen rays from the point *P* and the position of the eyeball in relation to the metallic marker and to the surface of the film (*F*).

geometric methods of localization and specifically in the modified Sweet method. It must, however, be remembered in examining this chart that the position of the foreign body has been indicated in reference to a model eye of a diameter of 24 mm. and that this is not necessarily the position of the foreign body within the eye of the patient. The actual eye may be larger or smaller than the model eye. It is more often smaller, because of trauma or partial collapse due to loss of aqueous. Therefore, if the foreign body appears to be outside of the mottled

globe on the localization chart it is probably outside the model globe. A greater fallacy is present when one assumes the foreign body to be actually in the eye because of its apparent position within the model eye as indicated on the localization chart.

Some assistance may be obtained by examining the eye with the ophthalmoscope or, if this is impossible, owing to hemorrhage, trauma or opacity of the media, the opposite eye may be examined. In this

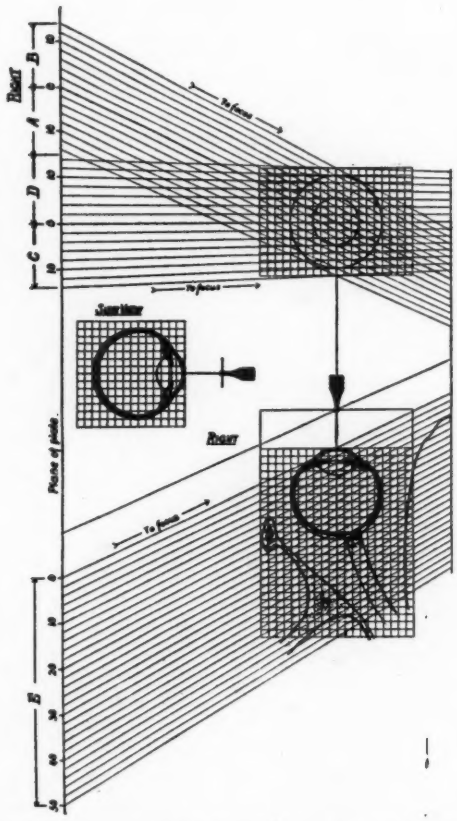

Fig. 7.—Chart used in the modified Sweet method of localization of a foreign body in the eye. These diagrams are typical of the geometric methods of localization. Entire localization is done in reference to the front and horizontal diagrams of the eyeball. The side view is not necessary but aids one to visualize the position of the foreign body.

manner one determines whether the eye is myopic or hyperopic, and this is an aid in deciding whether the actual eye is larger or smaller than the model eye.

The foreign body may, however, appear to be located within the globe on all three of the diagrams of the localization chart and yet

may be actually beyond the limits of the model globe.[6] This is possible because only the equatorial diameters are shown on the chart. As a means of clarifying this problem let it be supposed that, as illustrated in figure 8, an x-ray tube is pointed toward the pupil, the central beam being indicated by the arrow *b*. The image of the globe on the x-ray film *A* would appear circular, and the diameter of this circle would be equal to the greatest diameter of the globe. In this theoretical instance spreading of the roentgen rays is not considered, but they are thought

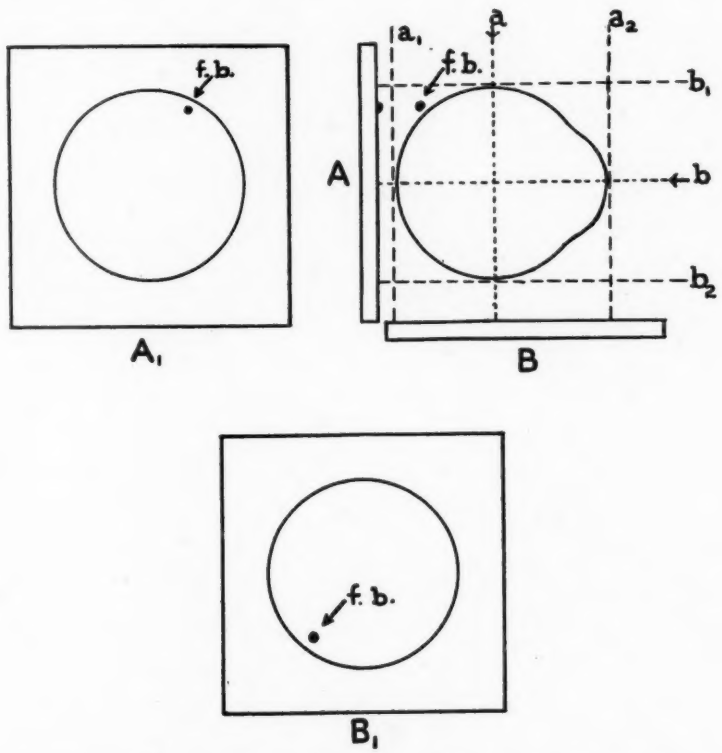

Fig. 8.—If a foreign body (*f.b.*) is beyond the limits of the globe, it may appear to be inside the globe in the anterior projection (*A*) if the beam of the roentgen rays *b* is directed as shown. A_1 would be the resulting image on the x-ray film. It would also appear on a film made at a right angle to the former (*B*), because the beam of the roentgen rays *a* projects the image of the globe as a circle equal to its greatest diameter, and B_1 would be the resulting image on the x-ray film.

of as a parallel beam of rays. If, therefore, a foreign body is in close relation to the posterior border of the sclera but not actually within the globe, it would be projected on the x-ray film A_1 as though

6. Stephenson, F. B.: Sweet Improved Eye Localizer Chart: How to Avoid Possible Misinterpretation, Am. J. Roentgenol. **16**:470 (Oct.) 1926

it were within the globe. By making a similar projection at an angle of 90 degrees it is possible to see that the foreign body could appear to be within the globe on both x-ray films and yet actually be outside, because the image of the eyeball has been on the x-ray film as the circle which shows only the greatest, or equatorial, diameter. In a similar manner it is easy to conceive that a third projection made in the other spatial plane might easily show a similar phenomenon. This is an actual error in many cases on the geometric localization chart. It may be argued that one does not make the projections for localization in this manner, but it must be fully understood and always remembered that the final result as reconstructed on the chart is visualized as though the projections of the roentgen rays were actually made in the three spatial planes, and the model globe is indicated as though it were actually visible. One must therefore be especially cautious in interpreting the appearance of a foreign body which seems to be in close relation to the posterior border of the sclera as within the globe, even though it may appear to be within the globe in all three diagrams on the chart. If the foreign body lies in close relation to one of the equatorial planes as indicated on the diagrams, there is less likelihood that the fallacy will enter, but when the foreign body appears to lie nearly midway between the equatorial planes one is likely to be led astray in assuming it to be within the globe. If one imagines the circles indicating the globe to be bounded by squares, the positions in which the greatest error is likely to occur are near the corners of these squares.

It is therefore of the utmost importance in this type of work to determine exactly the position of the foreign body in relation to the boundary of the sclera, since there is a greater likelihood that it lies definitely outside of the actual eye in those cases in which one can show it to be outside of the model eye. The manner of determining this important point will be presented next.

ACCESSORY METHODS

It has been shown that in using a geometric method of localization one is dealing with a model eye and localizing the foreign body in relation to the structures of this model. However, on the localization chart one views this model as though it were projected in the three equatorial diameters, and it is well to remember that the foreign body may appear within the model globe in all the diagrams on the chart and yet be actually outside this globe.

To ascertain if such is the case it is well to examine the position of the foreign body in relation to the diagram which illustrates the side view, or sagittal projection, of the globe. It is to be imagined that the diagram of the globe is enclosed in a square, the lines bisecting the

square overlying the horizontal and coronal equatorial planes. A foreign body lying in close relation to either the horizontal or the coronal equator is less likely to be beyond the limits of the model globe, but a foreign body which lies near the scleral boundary almost an equal distance between these equators is probably outside the limits of the globe. We are not so intimately concerned with those foreign bodies which lie in the regions of the anterior corners of the square, as the localization under discussion is considerably more accurate for a foreign body in the anterior hemisphere. Furthermore, if there is any doubt as to whether a foreign body which appears to lie in either of these positions is intra-ocular, a thorough search of the eyelids and the conjunctiva and soft tissue of the globe will in many cases settle the question. In the posterior positions, however, the question of double perforation arises and offers considerably more difficulty.

It remains to determine the actual plane in which the foreign body lies and to transfer this measurement to the appropriate diagram. This is done on the front view, or the coronal section. The procedure consists in bisecting the foreign body with a line extending to the scleral boundary. It may be done in either of two planes, horizontal or sagittal. If it is done in the horizontal plane, the measurement must be transferred to the horizontal projection; if it is done in the sagittal plane, the measurement must be transferred to the side view.

The measurement of the actual plane of the foreign body is then used as the diameter of a circle the center of which is the optic center of the globe. If the foreign body lies beyond the limits of this circle in either projection, it is outside the model globe. It is, of course, possible to transfer a similar measurement from any one projection to either of the others, but we use the front view as a routine to measure the planes as a matter of simplicity.

Figure 9 illustrates the method. Diameter *A* measures the actual width of the globe on a level with the foreign body in the horizontal plane. This is measured by calipers and transferred to the horizontal section as the diameter of a circle. The foreign body lies definitely beyond the boundary of the circle and is therefore extra-ocular.

Diameter *B* measures the actual width of the globe on a level with the foreign body in the sagittal plane. This is transferred in a similar manner to the side view, but the foreign body lies so close to the circle that one cannot be certain that it is extra-ocular. It must be remembered, however, that it is necessary for the foreign body to be outside the resulting circle in only one projection to be definitely beyond the limits of the model eye. In a similar manner, the diagnosis of an extra-ocular foreign body is made if the particle appears beyond the limits of the sclera on any one of the diagrams. The procedure just described appears to be simple and accurate, yet it is surprising

how many times in actual practice it will not definitely answer the question. In such cases one has a further check by mathematical means (fig. 10). The foreign body is known to lie a certain distance from each equatorial plane. *A* is the distance anterior or posterior to the coronal equatorial plane; *B*, the distance to the temporal or nasal side of the sagittal equatorial plane, and *C*, the distance above or

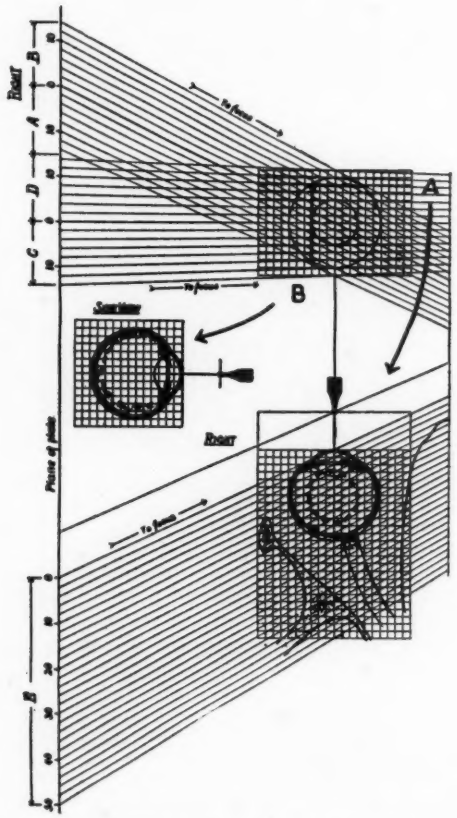

Fig. 9.—Method of transferring the actual diameter on which the foreign body lies to the appropriate diagram. Diameter *A* is parallel to the horizontal equatorial plane and must therefore be transferred to the horizontal view in the diagram. The result shows the foreign body to be definitely beyond the limits of the globe. Diameter *B* is parallel to the sagittal equatorial plane and must therefore be transferred to the side view. Judging by this result, there is no doubt that the foreign body is extra-ocular. Special care is not required in drawing these circles, as the entire procedure is intended only as an approximation.

below the horizontal equatorial plane. One then constructs in imagination a rectangular figure, the diagonal (*X*) of which would be the actual distance of the foreign body from the optic center. This is calculated by taking the square root of the sum of the three given

diameters according to the formula $X = \sqrt{A^2 + B^2 + C^2}$. The radius of the model eye is known to be 12 mm. Therefore, if the resulting figure for *X* is over 12, the particle is extra-ocular. In practice this is simplified by squaring the three diameters and adding them. If this gives more than the square of 12, or 144, the foreign body is extra-ocular; if it gives less, the foreign body must be intra-ocular.

The exact relation of the foreign body to the model eye has been determined, but this does not place the foreign body in anatomic rela-

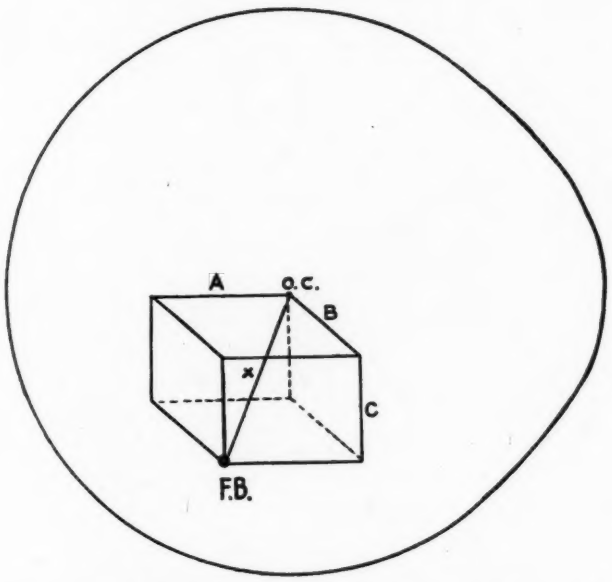

Fig. 10.—Diagram illustrating the method of determining by mathematical means whether a foreign body is intra-ocular or extra-ocular according to the formula $X = \sqrt{A^2 + B^2 + C^2}$. *O.C.* indicates the optic center; *F.B.*, the foreign body; *A*, the measurement posterior to the coronal plane; *B*, the measurement to the temporal or nasal side of the sagittal plane; *C*, the measurement below the horizontal plane, and *X*, the diagonal of the rectangular parallelopiped.

tion to the eye of the patient. To accomplish this two procedures are used—bone-free roentgenography of the anterior hemisphere and the parallax method.

If the foreign body appears to be in the anterior hemisphere and cannot be seen on ophthalmoscopic or slit lamp examination, it may often be shown by a bone-free projection.[7] This is made by bending a dental film and slipping it between the eyelids and the bony orbital

7. Butler, T. H.: Bone-Free Radiographs, Proc. Roy. Soc. Med. (Sect. Ophth.) **17**:6, 1923-1924. Lindblom, K.: Bone-Free Radiography of the Eye, Acta radiol. **15**:615, 1934.

margin (fig. 11 *A*). The beam of the roentgen rays is then directed at an angle of 90 degrees or slightly greater toward the film, the central ray being aimed at the outer canthus. A very soft radiation is used, and the film (fig. 11 *B*) shows the cornea and anterior portion of the globe between the lids. In this manner assistance is given in ascertaining the position of the particle and in recognizing some particles which are so small or have a specific gravity so low that they cannot be seen on the usual projections. It is essential to have the lids opened as widely as possible to show the globe to advantage. We have in a few cases recognized a foreign body buried in the eyelid by this means. Some workers trim down the x-ray film and slip it

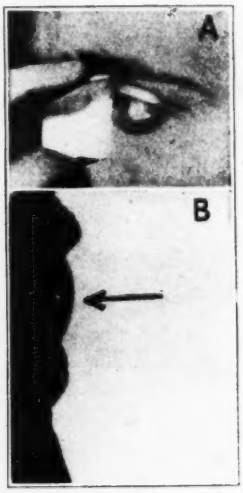

Fig. 11.—*A*, method of holding a dental film for soft tissue projection of the anterior portion of the globe. It is essential to have the eyelids separated as widely as possible. In practice an assistant holds the eyelids apart. *B*, roentgenogram taken by the method of soft tissue projection, showing the foreign body.

into the conjunctival sac; others make similar projections from above, but we have not found that these practices add anything essential and believe that projection with the use of a dental film is sufficient for all routine work.

The parallax method [8] is based on the principle that if a foreign body is intra-ocular it will rotate about the optic center when the eye is directed between predetermined external points of visual fixation. Thus the rotation of the eye becomes the factor of importance in determining whether a particle apparently within the model eye is also in the actual eye or is outside.

8. Verwey, A.: Localization of Foreign Bodies in the Eye in Relation to the Rotation Center, Am. J. Ophth. **7**:337, 1924.

The patient is placed with the side of the head against the x-ray film and directed to gaze at a spot on a level with the eye. A film is exposed, and another film is placed in position without the position of the head being disturbed. This requires some sort of film-holding device. The patient is instructed to look at a point above the first position by rotating his eyes upward but is cautioned not to change the position of his head. Another film is exposed, and a third is placed in position. The patient then rotates the eye downward to a point an equal distance below the horizontal meridian, and a third film is exposed. In routine practice we make three heavy marks on a yard-stick, the upper and lower points being equal distances from the

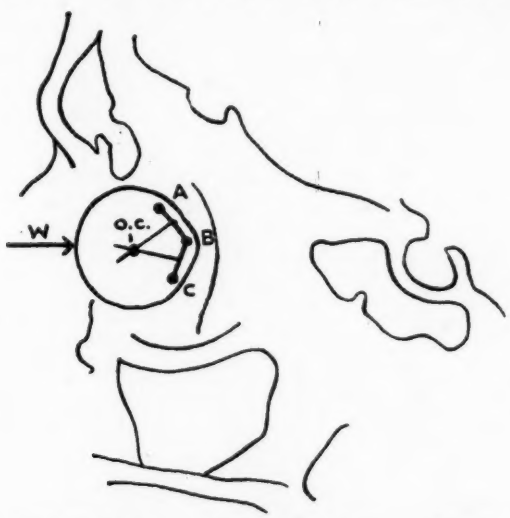

Fig. 12.—Tracing made by superimposing the x-ray films. *O.C.* indicates the optic center; *A, B* and *C,* images of the foreign body as the eyeball rotates between the points of visual fixation, and *W,* wire indicating the optic axis and the tangential plane of the cornea.

midpoint. This is supported about 4 feet (1.2 meters) in front of the patient, with the middle marker on a level with the eyes.

One now has three films which, after being processed and dried, may be superimposed. There will be three images of the foreign body present, if it has moved owing to the rotation of the globe. One makes a tracing of the films by superimposing a sheet of thin paper and drawing the main anatomic marks and the image or images of the foreign particle and adds to this the approximate position of the globe. This is done by placing across the bridge of the nose, before the films are exposed, a straight fine wire, held on by adhesive tape in such a manner that the end of the wire is adjacent to the tangential plane of the cornea and centered at the midpoint of the pupil as judged from the lateral aspect (fig. 12). This wire is shown on all three roent-

genograms and not only insures accuracy in superimposing but indicates the tangential plane of the cornea and the level of the optic axis. The eyeball is now roughly sketched in, and one has a drawing similar to figure 12. The optic center can be indicated with reasonable certainty, and it may be determined whether the particle rotates about this center by connecting the three images of the foreign body with straight lines and dropping ordinates from these lines. If the ordinates cross at or near the optic center, the foreign body is intra-ocular; if

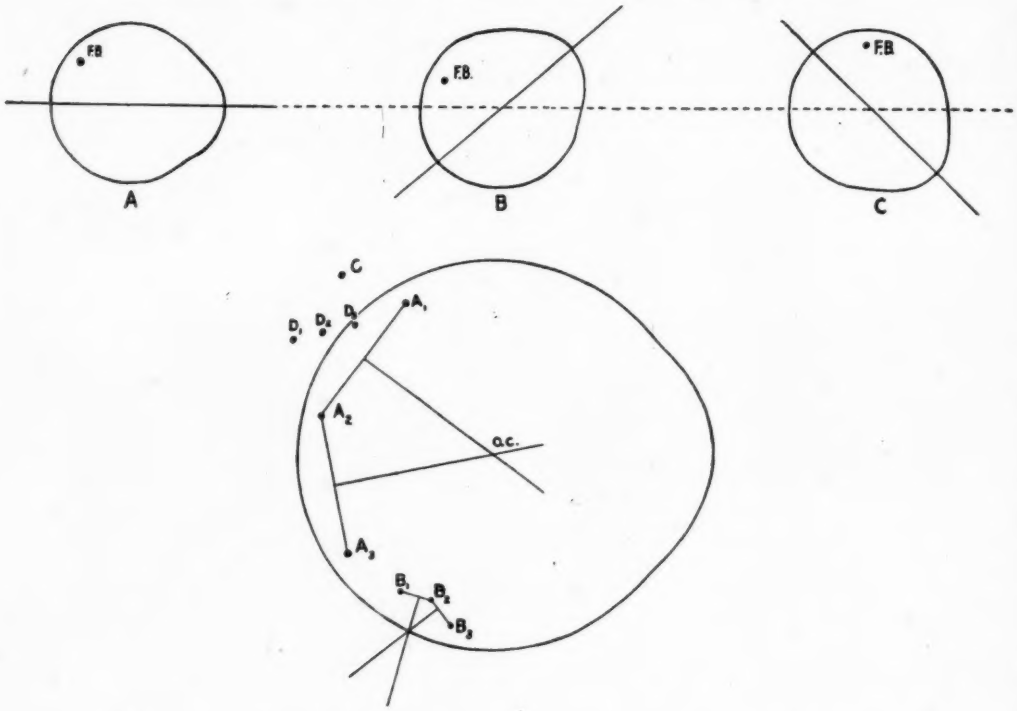

Fig. 13.—*A*, position of a foreign body (*F.B.*) in the globe with the eyeball directed to the horizontal point; *B*, position with the eyeball rotated upward; *C*, position with the eyeball rotated downward. A_1, A_2 and A_3 show the behavior of an intra-ocular foreign body; B_1, B_2 and B_3, the movement of an extra-ocular foreign body (*C*), and D_1,D_2 and D_3, the behavior of a foreign body which has become embedded in the superior rectus muscle.

they cross at any other point either within or outside of the globe, the particle is extra-ocular. This is illustrated in figure 13, which shows the behavior of both types of bodies.

In the great majority of cases of an extra-ocular particle the movement is nil or so slight that there is no question as to the position of the particle. Two sources of error may be present. If a body that is extra-ocular is embedded in Tenon's capsule or is adherent to the

posterior part of the sclera it may behave as though it were within the eye, and a foreign body in an unusually fluid vitreous may behave as though it were extra-ocular. Thus it may be seen that all problems of localization of an intra-ocular foreign body cannot be accurately settled by this means, but it is of great advantage in those cases in which the particle is in close relation to the posterior part of the sclera. I have previously stated that localization by geometric methods is less accurate in the posterior portion of the globe, and the ability to localize a foreign body here is the greatest advantage of the so-called parallax method as a check-up procedure.

Another possible problem may arise in cases in which it is necessary to decide whether the foreign body is intra-ocular when it lies near the lateral poles of the eye and does not show sufficient rotation about the axis for localization by the method described, owing to its close proximity. We have in cases of this condition made postero-anterior projections in a similar manner to that described, the eyes being rotated laterally.

Thus many problems arise in the routine localization of intra-orbital foreign bodies which require various methods supplementary to the standard geometric procedures. It is possible to determine with considerable accuracy the exact position of the particle in relation to the structures of the patient's eye. In a few cases there still remains a doubt, and further check-up studies are required. These problems will be discussed in the final section.

VISIBILITY OF FOREIGN BODIES

It is important for the ophthalmologist to familiarize himself with the methods of the roentgenologist, but it is equally advantageous for the roentgenologist to be acquainted with the problems as they present themselves to the operating ophthalmologist. If a foreign body is responsive to the magnet, the operator will usually try to extract it through the wound made by its entrance. If the foreign body is in the vitreous, it is of great importance in many instances to make a check-up localization to determine how far it has been displaced, if the operator has not succeeded in removing it. If it cannot be drawn through the ciliary body or if it adheres to the posterior capsule of an undamaged lens, it is often better to do a sclerotomy rather than continue to use magnetic traction in the original direction. If it is embedded in, or becomes firmly fixed to, the iris, it is often better to do an iridectomy. If the foreign body is large and is in the vitreous, especially in a case in which the lens is clear or the condition has been of long standing, with organization of material about the particle, it may often be extracted through a posterior sclerotomy. If

it is in the lens, noninterference is sometimes the best policy until cataract develops. If the particle seems to be buried in the sclera and the eye is quiet, many operators do not disturb it. Others give a single trial with the magnet and send back the patient for relocalization of the foreign body. If there is no movement, they go no further. If there is a double perforation and the eye is quiet, the foreign body is usually not disturbed. If the body is nonmagnetic, it may sometimes be removed by searching blindly and sometimes by biplane fluoroscopic control. If it is not removed, it is a potential source of danger not only to the injured eye but also to the good eye, and if there is any doubt, enucleation should be seriously considered.

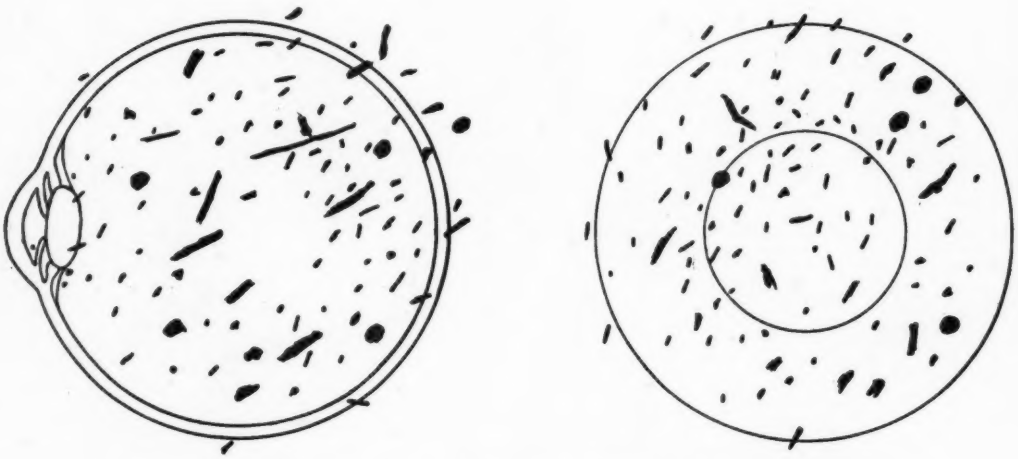

Fig. 14.—Relative size and position of the intra-orbital foreign bodies in one hundred unselected cases, as shown by geometric localization. There might be a question of double perforation in from twenty to twenty-five of these cases.

We recommend that all roentgenologists be familiar with the simple problems just mentioned and that they follow the patient when possible or at least learn the manner and results of the operative procedure. This will greatly aid them in acquiring a clearer understanding of the problems involved. I have shown diagrammatically (fig. 14) the relative size and position of the particles in one hundred unselected cases observed in the roentgen department at the Wills Hospital without reference to which eye was involved. The drawings illustrate the position as shown on the localization charts. It will be noted that there arises a possible question of double perforation in about twenty to twenty-five of the cases. It appears that in one case the foreign body was buried in the posterior part of the sclera. Actually many of these eyes were so badly infected that enucleation was the procedure of choice. I would estimate that in at least 10 per cent of the routine cases this

question of double perforation has arisen. In nine of the ten cases the question can be settled by the previously described procedures, but there still remain a few cases in which the condition is puzzling in spite of all attempts to obtain complete evidence. In these cases we resort to the injection of air into the capsule of Tenon and auto-visualization.

The injection of air into Tenon's capsule is made in an attempt to render the posterior hemisphere visible on roentgen examination. Some workers have used opaque material, but we have had such good results with air that we believe it to be preferable. Iodized poppy-seed oil 40 per cent, diodrast and other opaque mediums remain for a long period after injection and tend to prevent clear visualization of the foreign particle. Air appears to diffuse or absorb quickly, has been perfectly safe and harmless thus far and aids rather than prevents visualization of the particle. We do not think that sufficient evidence has been gathered as yet to recommend the practice promiscuously, and it should never be attempted by the radiologist alone. However, when the question of double perforation arises and cannot be settled by other means, especially if judgment as to an operation hinges on the result, we have not hesitated to use the following method.[9]

The eye is thoroughly anesthetized. The patient is then instructed to gaze downward and inward, turning the injured eye toward the tip of the nose. A point about midway between the superior oblique muscle and the external rectus muscle is chosen, and the eye is fixed in position. A needle is bent at an angle of about 45 degrees and inserted below the conjunctiva about 4 mm. anterior to where the reflection of Tenon's capsule is thought to be. It is now introduced posteriorly from 3 to 4 mm. under the conjunctiva until it is sufficiently far back to be over the capsule (fig. 15). By rotation the point is turned downward, and the capsule is punctured. It is essential to bend the needle and proceed by the technic just described, because the air injected would run directly back about the barrel of a straight needle and escape. A syringe containing from 8 to 10 cc. of air is then attached, and the injection is made gently but steadily. When about 6 cc. has entered, there are an increase in resistance and a slight but distinct proptosis. The proptosis is a guide to the correctness of the procedure. If the needle is not in Tenon's space the injected air will cause a bulge in the conjunctiva without proptosis. It is best in this case to discontinue the injection for about forty-eight hours and then try the procedure again. The patient should complain of little or no discomfort.

9. Spackman, E. W.: X-Ray Diagnosis of Double Perforation of the Eyeball After Injection of Air into the Space of Tenon, Am. J. Ophth. **15**:1007 (Nov.) 1932.

X-ray films are exposed at leisure, and the processing is done immediately to be sure that the results are satisfactory. It is a wise precaution to make a single lateral projection before the injection to compare with subsequent projections. The air in Tenon's space may form a contrast layer (fig. 16*A*) or a sharply demarcated band (fig. 16*B*), which should be readily distinguished on the film. It is advisable to make exposures both with and without the use of the Potter-Bucky diaphragm, but casettes and intensifying screens should always be used. A single lateral projection is not to be depended on, unless the foreign particle is definitely seen to be extra-ocular. The same error can be easily made as described in discussing the charts used in the geometric methods of localization, and the foreign body appears to be within the globe because of superimposition. There must be a sufficient number of exposures made from various angles so that the

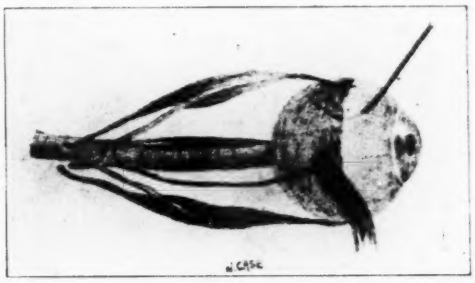

Fig. 15.—Drawing illustrating the method of injecting air into Tenon's capsule. The bent needle is inserted from 3 to 4 mm. under the conjunctiva before penetrating Tenon's capsule. By going through two layers in this manner there is less likelihood that the injected air will escape.

beam of the roentgen rays meets the posterior sclera tangentially at the point where the foreign particle lies. This is done by gradually rotating the head and making serial exposures after each slight movement. The direction of rotation can be determined by examining the localization chart and by bearing in mind the fact that the image of the particle appears to approach the scleral edge after rotation if the rotation is in the right direction.

By this method a final diagnosis of double perforation may be verified or disproved. This procedure is not successful in all cases, however. Occasionally an extremely nervous patient will cause difficulty, and in the case of other patients we found that injection was not possible. We believe that in cases in which the latter was true the difficulty was due to adhesions or fusion between the capsular layers.

The patient is comfortable afterward, and the air is completely absorbed in from two to four days.

One other method of check-up is used and is being developed at present, but too few data are as yet available to enable one to be certain of its value. This we call autolocalization. Pirie [10] found that after complete relaxation of accommodation it was possible to visualize directly the roentgen rays without aid. Contrary to general belief, the retina is somewhat sensitive to the wavelength of the roentgen rays. I therefore place the patient before the x-ray tube in a darkened room and ask him whether he can see a black spot within the greenish fluorescent light which is visible when the tube is energized. If so,

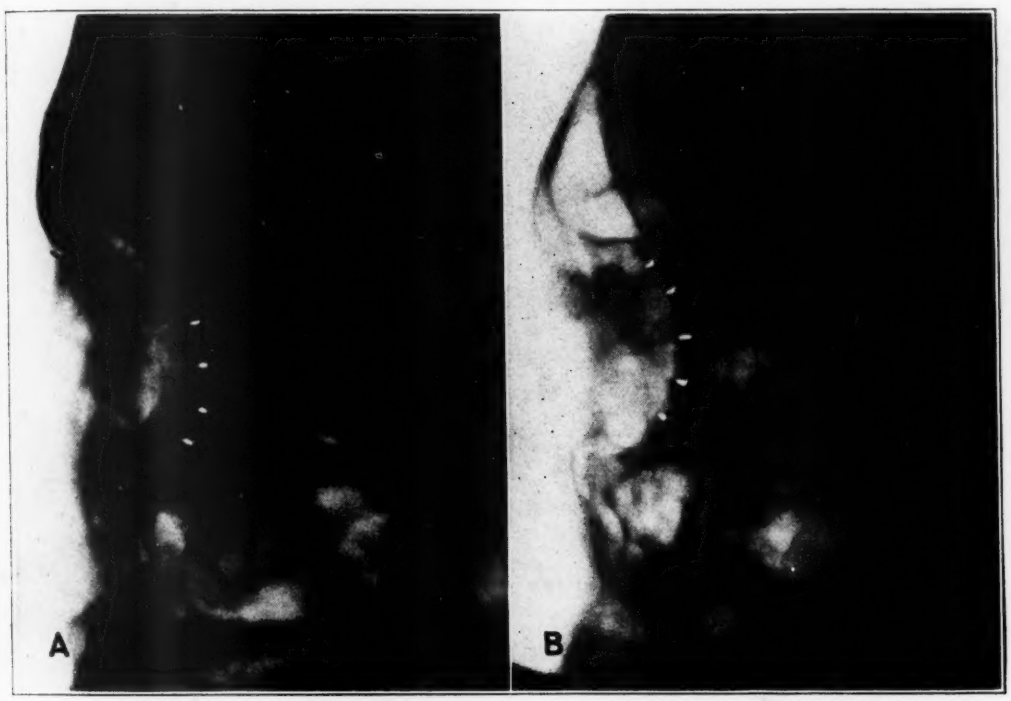

Fig. 16.—Sometimes the air causes a zone of contrast (*A*), and at other times it causes a band of decreased density (*B*). In both the cases illustrated the foreign body is shown to be extra-ocular.

he is asked to describe it and to draw its relation to cross-wires which are placed in a frame before the injured eye. A very narrow beam is used so that no more exposure than is absolutely necessary is given. The patient is then asked to draw a diagram of what he saw, placing the spot in relation to the cross-wires. He will always place it in the quadrant diametrically opposite that where it actually is, since there is no reversing action of the lens to the roentgen rays but it is

10. Pirie, A. H.: An Apparatus for Reading with Closed Eyes, Brit. J. Radiol. **7**:111 (Feb.) 1934.

interpreted as though it were reversed, as is usual with the beam of light. I have apparently shown thus far that the foreign body, if small, must be within a few millimeters of the retina to be seen and that it can be placed by the patient with considerable accuracy, which helps to check the data for localization. In cases in which the foreign body has perforated the retina, we have failed to obtain results with this procedure, and therefore we believe that this helped us in several instances in which the foreign body had perforated or was buried in the sclera. Between thirty and forty patients have been tested in this manner, and this method is mentioned for the benefit of those workers who have the opportunity to try it. Complete relaxation of accom-

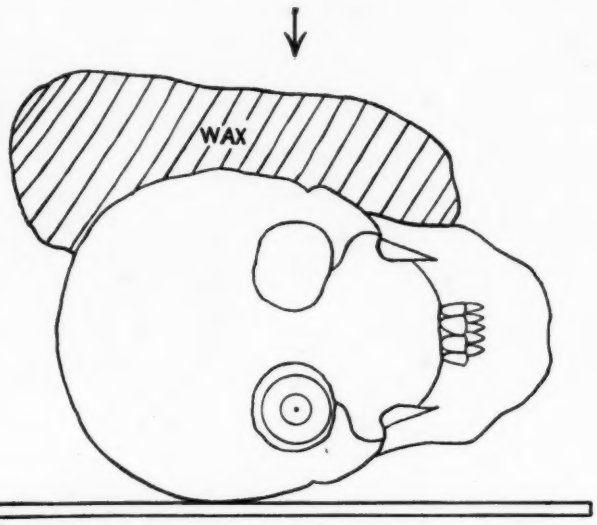

Fig. 17.—Method of approximating the density of the human head as noted roentgenographically by using a dried skull and wax. An enucleated eye was placed within the skull, and wax was added until experimentally a roentgenographic effect was obtained similar to that obtained with the average human head.

modation is essential; the patient must remain in total darkness between fifteen and twenty minutes before the attempt is made. Warning must also be given to avoid overexposure, as it is easy to forget this precaution.

We have sought to show experimentally the relation between the size, the specific gravity and the density of the tissue as applied to the problem of the visualization of intra-orbital foreign bodies. It has long been recognized that the roentgen visibility of a body is directly proportional to the specific gravity. We therefore collected samples of all materials which have been found to act as intra-ocular foreign particles in the cases observed by us during the past twelve years and proceeded empirically to build a model of actual working conditions (fig. 17). A dried skull was coated on one side with paraffin until

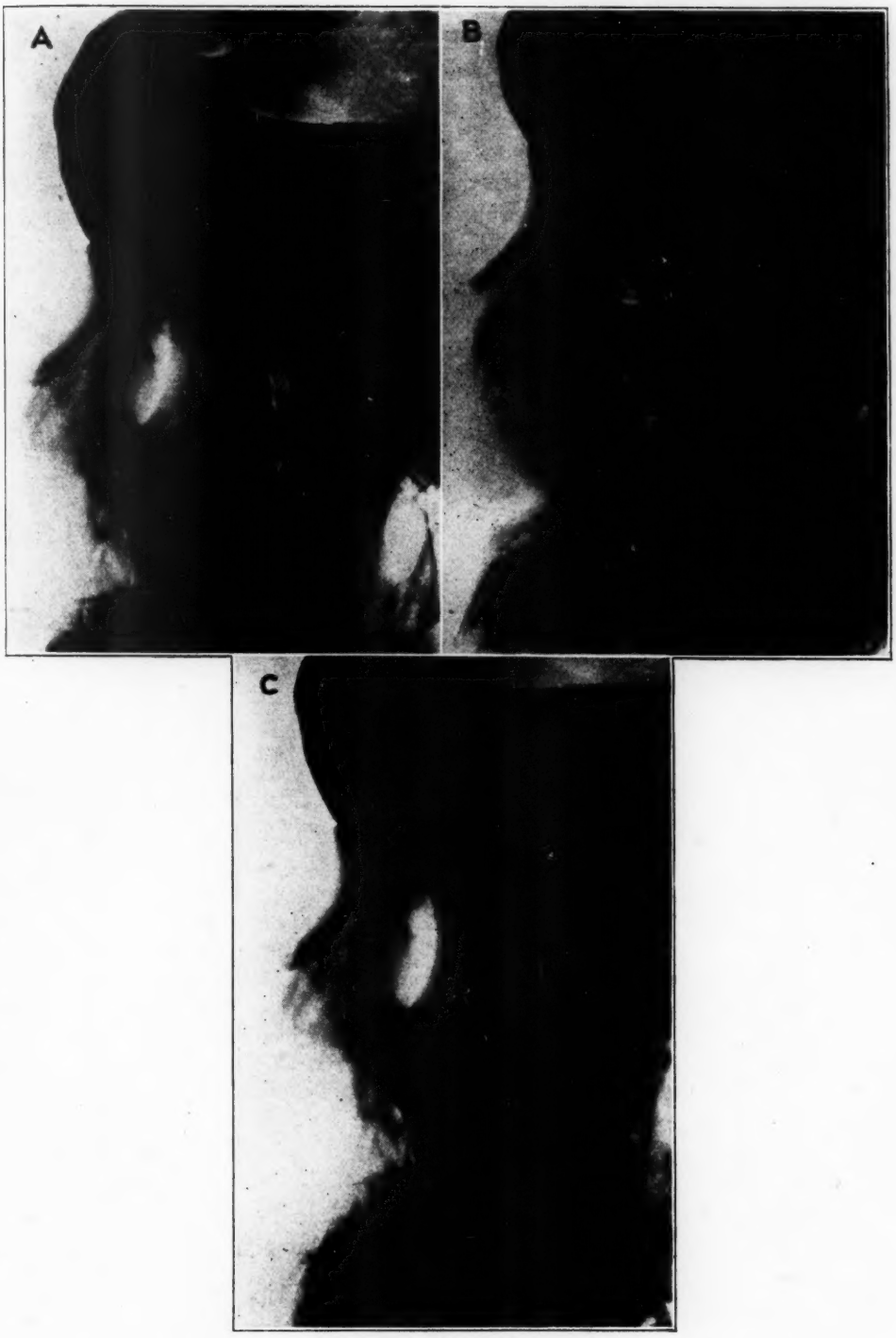

Fig. 18.—*A*, roentgenogram taken after a particle of glass 1.5 mm. in diameter, with a specific gravity of 3, was embedded in an eye which was placed in the skull. This is not visible on the x-ray film. *B*, roentgenogram taken after a particle of aluminum 2 mm. in diameter, with a specific gravity of 2.5, was embedded in a similar manner. This is barely visible through one layer of bone. *C*, roentgenogram taken after a particle of marble 3 mm. in diameter, with a specific gravity of 2.8, was embedded. This is visible through one layer of bone.

lateral exposure through it gave a roentgenogram showing the same average quality, detail and density that are usually obtained with the head of a living person. The paraffin afforded approximately the same degree of absorption and scattering as human tissue. An enucleated eye was then placed in the orbit, and we proceeded to embed within the eye particles of the various substances. These were chosen by crushing the material and obtaining roughly spherical bits which ranged in size on measurement with a magnifying glass and calipers from 0.5 mm. upward in steps of 0.5 mm. These were sorted, arranged and tested in the manner already described, the larger particles being tested first, until the limit of visibility through the orbital bones was reached (fig. 18). This limit was noted and checked, and further studies were carried out by embedding the particles within the eyeball after its

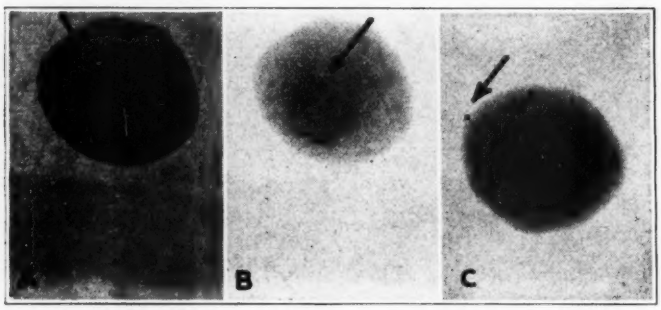

Fig. 19.—Results obtained by placing the enucleated eye on the x-ray film and embedding a foreign body within it. These are similar to those obtained with bone-free roentgenograms. *A*, roentgenogram showing a particle of lead less than 0.5 mm. in diameter, with a specific gravity of 11.3. This was deeply embedded and exposed to fairly dense radiation. *B*, roentgenogram showing a particle of steel less than 0.5 mm. in diameter, with a specific gravity of 8.5. This was deeply embedded, and radiation of lesser density was used. *C*, roentgenogram showing two particles of emery less than 0.5 mm. in diameter, with a specific gravity of 4, one of which was placed on the surface of the eye and the other embedded. The embedded particle is barely visible on the film.

removal from the skull. In this manner we obtained data which gave us the approximate size a foreign body of a given specific gravity must be to be visualized by the usual method of localization and also by the bone-free projection (fig. 19). We noted the results as: (*a*) visible through bone, (*b*) questionably visible through bone, (*c*) visible through soft tissue and (*d*) questionably visible through soft tissue. It is realized that this is only a relatively rough guide, as superimposition of bony layers in the orbit would obscure some particle which would be plainly visible in other positions. However, it acts as a

fair guide and has been found to be accurate during the past few years. By referring to the chart the ophthalmologist may obtain some idea of the required size of an intra-orbital foreign body before it can be shown on roentgen examination. He may often estimate to a certain extent the probable size of the foreign body by the patient's statement, the size of the wound caused by the entrance of the foreign body and other evidence and obtain more accurate assistance from the report

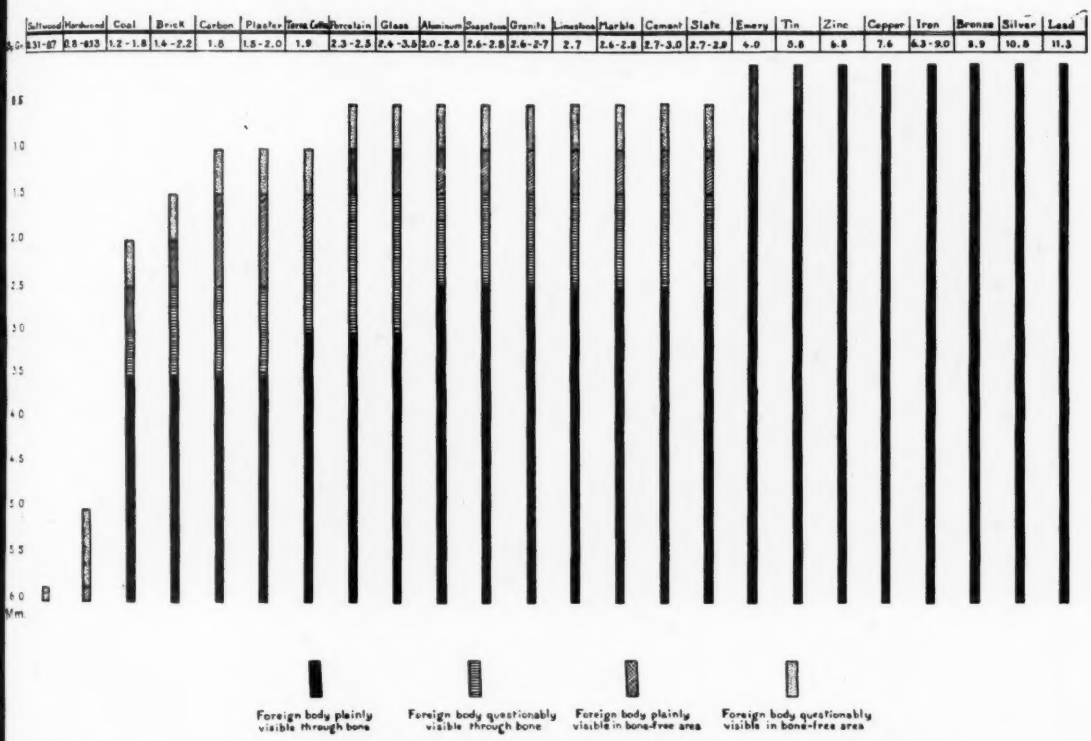

Fig. 20.—Composite chart compiled from data obtained in the experimental work described in the text, showing the degree of visibility of various particles in relation to their size and specific gravity.

of the radiologist. This is of special service in the case of particles of nonmagnetic substances. The chart (fig. 20) is a handy means of referring to the aforementioned data, and we have found it of value in many problems. In making the experiments we sought to duplicate exactly the conditions under which the work on localization is done relative to the distance of the tube from the film, the focal spot, the exposure, etc., so that it would be of assistance in checking the routine work.

SUMMARY

In regard to the localization of an intra-orbital foreign body, the roentgenologist should always familiarize himself with the history, examine the wound made by the entrance of the foreign body and obtain any other pertinent data bearing on the case. Roentgenograms for identification are sometimes taken if there is a question of lodgment in some structure outside the orbit. Special projections of the orbital apexes and the optic canal are often useful. The usual study for localization is then made, invariably supplemented by soft tissue projections if the foreign body is thought to be minute or of low specific gravity and cannot be located on the usual films. We cannot too strongly stress the importance of perfect immobilization, as very slight movements of the globe may completely invalidate the results. If the results of localization are not perfectly satisfactory, a check-up study is made.

The chart used to record the results of the geometric method of localization is examined, and if any doubt exists, geometric and mathematical measurements are made to ascertain whether the foreign body is within the model eye. The parallax study is resorted to in nearly all cases in which the particle is in the vicinity of the posterior part of the sclera. If doubt still remains and it is important to determine whether double perforation is present, roentgenography after the injection of air into Tenon's capsule and autovisualization are attempted. Reliance is not placed on any one study, but all must be considered together. The report to the operating ophthalmologist includes a description and the results of all the studies made and the judgment of the roentgenologist in interpreting the data obtained.

ISOLATION OF VERHOEFF'S LEPTOTHRIX IN A CASE OF PARINAUD'S SYNDROME

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The type of the so-called clinical syndrome of Parinaud which my associates and I have seen most frequently is undoubtedly that which Verhoeff showed to be due to an infection with leptothrix and which might reasonably be excluded from syndromal grouping and referred to as a separate entity by a name of its own, such as "leptothrix conjunctivitis of Verhoeff." Although the literature gives tuberculosis of the conjunctiva a place in this group, it ought not to be difficult clinically to differentiate between the two conditions. In leptothrix conjunctivitis of Verhoeff there are an acute febrile onset associated with the conjunctival inflammation and a rapid enlargement of the preauricular, parotid and submaxillary glands. The palpebral conjunctiva shows marked swelling—a solid swelling due to cellular infiltration—and is intensely red, but the bulbar membrane shows a relatively scanty discharge or chemosis. The surface varies from a finely granular one with regular folds due to the swelling to a nodular or papilliform surface. The characteristic small semiopaque areas seen here and there look as if cooked with the point of a diathermy needle. The signs and symptoms persist for a fortnight or so and then slowly subside, till normality is restored in five or six weeks. The fever shows a rise in the evening and may persist for ten days or more. The blood picture may be normal or may show leukocytosis. The cutaneous tuberculin test and the Wassermann test may be positive or negative and are therefore of little help. Although the clinical course of this disease is fairly straightforward, in certain cases it presents difficulties of clinical diagnosis to those who have not had experience in its vagaries and even to those who have. My associates and I were pleased on this account to be able to apply a relatively simple diagnostic method in a case recently observed, namely, the cultivation of Verhoeff's leptothrix from the fluid of the preauricular gland. It is by no means simple to obtain histopathologic preparations which show the organisms in the gray areas of the conjunctiva, and unless one's laboratory technic is good, failure is likely. Dr. Verhoeff sent me some of his histopathologic specimens some years ago, and with his technic positive results were obtained in the typical clinical condition. This method is, however, too

difficult for use in the laboratory of the ordinary ophthalmic hospital; the cultural method is simpler. Neither method is certain, but either is as satisfactory as, say, inoculation of animals with a portion of the affected tissue in tests for tuberculosis or a Wassermann test for

Photomicrograph of slide preparation from a pure culture of Verhoeff's leptothrix taken by Lieutenant-Colonel H. E. Shortt.

syphilis. The material was obtained from the preauricular gland in the usual way for obtaining glandular fluid under strict bacteriologic precautions as to sterility, with a needle mounted on a syringe containing a little broth (0.5 cc.). The point of the needle was moved about in the gland to a slight extent, while positive and

negative pressure were being gently made with the syringe. The cultures were made immediately by Dr. B. P. B. Naidu, of the Medical Research Department of the Government of India (Indian Research Fund Association), attached to the King Institute of Preventive Medicine, Guindy, Madras, who at the time was carrying out research work in this hospital in collaboration with me (on trachoma and in other fields). The following details were supplied by him:

METHOD

The patient was G. a girl aged 16. Material for culture was aspirated from the center of the preauricular gland by a syringe, under sterile conditions. The material was inoculated onto the surface of (1) Soparkar's heated human blood agar, (2) hydrocele agar and (3) ordinary agar enriched by a surface smear with fresh human blood. The tubes were incubated at 30 C. anaerobically. After forty-eight hours of incubation the culture tubes showed discrete colonies, a few being present on each of the three culture mediums used.

Growth was obtained in subcultures under both aerobic and anaerobic conditions; the growth was more luxuriant under anaerobic cultivation.

Smears stained by Gram's method showed a gram-positive leptothrix. Subcultures were made on Soparkar's heated human blood agar (on this medium the growth tends to colony formation). On Dorset's egg medium the growth is profuse, producing pitting and lysis; on agar the growth is orange, moist and shining. In peptone water the growth produces general turbidity, with a heavy deposit; similar characteristics are observed in nutrient broth; in dextrose broth the growth is scanty. The appearance of the colony on agar is circular and raised, with an irregular wavy fringe which is more transparent than the colony. The growth produces acidity in dextrose, saccharose, salicin and maltose and no change in lactose, dulcitol and mannitol. Other sugars were not tried. In milk it produces alkalinity.

MEDIUMS

Soparkar's medium (for the growth of influenza bacilli) was described in the *Indian Journal of Medical Research*.[1] For the convenience of those interested in bacteriology the mediums used are detailed.

Ordinary Nutrient Agar Mixed with a Preparation of Human Blood.—A medium containing 3 per cent nutrient agar is prepared according to the following formula:

Meat extract	1 liter
Peptone	20 Gm.
Sodium chloride	10 Gm.
Agar	30 Gm.

The reaction is rendered slightly alkaline to litmus by the addition of sodium carbonate. The meat extract is prepared by boiling 1 Kg. of meat with 2 liters of water for forty minutes.

1. Indian J. M. Research **6**:418, 1918-1919.

Soparkar's Medium.—The preparation of the human blood which is added to the agar is made as follows: One volume of freshly drawn human blood is added to a flask containing four volumes of sterile citrated saline solution (0.5 sodium citrate and 0.85 per cent sodium chloride). The blood and saline solution are thoroughly mixed by shaking, and the fluid is then heated in a water bath at 66 C. for thirty minutes. The heating results in the formation of a fine brown precipitate, which is removed by filtration through sterilized filter paper. All precautions should be taken to prevent contamination. The filtrate is a clear, deep red fluid, and 1 cc. of it is added to 10 cc. of melted agar at 50 C. After mixing, the tubes are sloped and allowed to set. The medium thus prepared is clear and transparent.

Hydrocele Agar.—Fifty cubic centimeters of sterile hydrocele fluid is added to 10 cc. of melted 4 per cent nutrient agar at 48 C. The ingredients are gently mixed, and the tubes are sloped; vigorous shaking should be avoided.

Other Mediums.—The ascitic fluid agar of Morax, which gives excellent growths for many of the conjunctival pathogens in Paris, was not so useful in India, since the ascitic fluid locally available had not an optimum albumin content (from 1.5 to 1.6 per cent of the albumin content of the blood serum), nor could the "ox coagulated serum," which Morax considers an excellent medium, be conveniently obtained.

Comment.—Dr. Naidu found that with the three mediums, the formulas of which have just been given, good all-round results were obtained with the conjunctival flora, on both aerobic and anaerobic cultivation.

RELATIONSHIPS BETWEEN ANISEIKONIA AND AMETROPIA

FROM A STATISTICAL STUDY OF CLINICAL CASES

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AND

L. F. MADIGAN, M.S.

HANOVER, N. H.

INTRODUCTION

The importance of aniseikonia as an etiologic factor in certain visual disorders has been proved by previous investigations,[1] but there are still many misconceptions [2] concerning the relationships between aniseikonia and ametropia. The object of this paper is to clarify these misconceptions by presenting a statistical analysis of the cases of 283 patients who have received definite relief from ocular discomfort by the correction of aniseikonia.

During the early years of the development of the research on aniseikonia the patients who were examined at The Eye Clinic of the Dartmouth Medical School constituted what might be called a highly selected group.[1a] At least 90 per cent of them were referred to The Eye Clinic because all other procedures used had failed to give relief or reveal any organic cause for their discomfort. In general, the symptoms

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From The Eye Clinic of the Department of Research in Physiological Optics, Dartmouth Medical School.

The clinical data of this paper were presented at the three hundred and eleventh meeting of the New England Ophthalmological Society, at Hanover, N. H., May 23, 1936.

1. (*a*) Carleton, E. H., and Madigan, L. F.: Size and Shape of Ocular Images: II. Clinical Significance, Arch. Ophth. **7**:720-738 (May) 1932. (*b*) Madigan, L. F., and Carleton, E. H.: A Clinical Report of the Correction of Differences in the Size and Shape of Ocular Images, Ann. Distinguished Serv. Fund. Optom. **1**:71-97, 1932. (*c*) Hughes, Wendell L.: Aniseikonia: Some Clinical Observations, Am. J. Ophth. **18**:607-615 (July) 1935; (*d*) Aniseikonia, ibid. **19**:686-688 (Aug.) 1936. (*e*) Doane, H. C.: The Clinical Significance of Differences in the Relative Size and Shape of Ocular Images, Tr. Am. Acad. Optom. **8**:117-127, 1933; (*f*) Clinical Developments and Observations in the Correction of Aniseikonia, ibid. **9**:31-43, 1935.

2. Jackson, Edward: The Importance of Aniseikonia, Tr. Sect. Ophth., A. M. A., 1936, p. 27-48. Friedenwald, J. S.: Diagnosis and Treatment of Anisophoria, Arch. Ophth. **15**:283-307 (Feb.) 1936. Ludvigh, E. J.: Aniseikonia, Am. J. Ophth. **19**:292-301, 1936.

reported by these patients included functional disturbances, such as asthenopia, photophobia and disability in reading; local ocular disturbances, such as aching and burning of the eyes and blurring of print, and neurologic disturbances, such as headaches, nervousness and general fatigue. A large percentage of the patients obtained remarkable relief from their ocular discomfort when their aniseikonia had been corrected. As is to be expected in such a group, there were also many patients who did not obtain relief. The fact, however, that so many of the patients had a beneficial reaction from the treatment indicated the clinical value of investigating this anomaly as a possible cause of some of the disturbances mentioned.

A statistical study of the patients treated at The Eye Clinic was carried out during the past six years, during which time 829 patients

TABLE 1.—*Classification of Two Hundred and Eighty-Three Clinical Cases in Which Aniseikonia Had Been Corrected and in Which Definite Relief from Ocular Disturbances Was Reported*

	Number of Cases	Percentage
(a) Changes in the refractive corrections made at the time of correcting the aniseikonia		
Refractive correction not changed	38	13
Refractive correction changed less than 0.50 D.	65	23
Refractive correction changed more than 0.50 D.	68	24
No classification as to refractive changes	112	40
(b) Magnitude of refractive errors		
Refractive errors of both eyes less than 0.50 D.	31	11
Refractive errors of both eyes between 0.50 and 1.00 D.	32	11
Emmetropia	14	5
(c) Refractive condition of the two eyes		
Bilateral emmetropia	14	5
Isometropia	74	26
Anisometropia of 0.25 to 0.50 D.	76	27
Anisometropia of 0.50 D. or more	119	42
(d) Prisms removed when the correction for aniseikonia was given	19	6

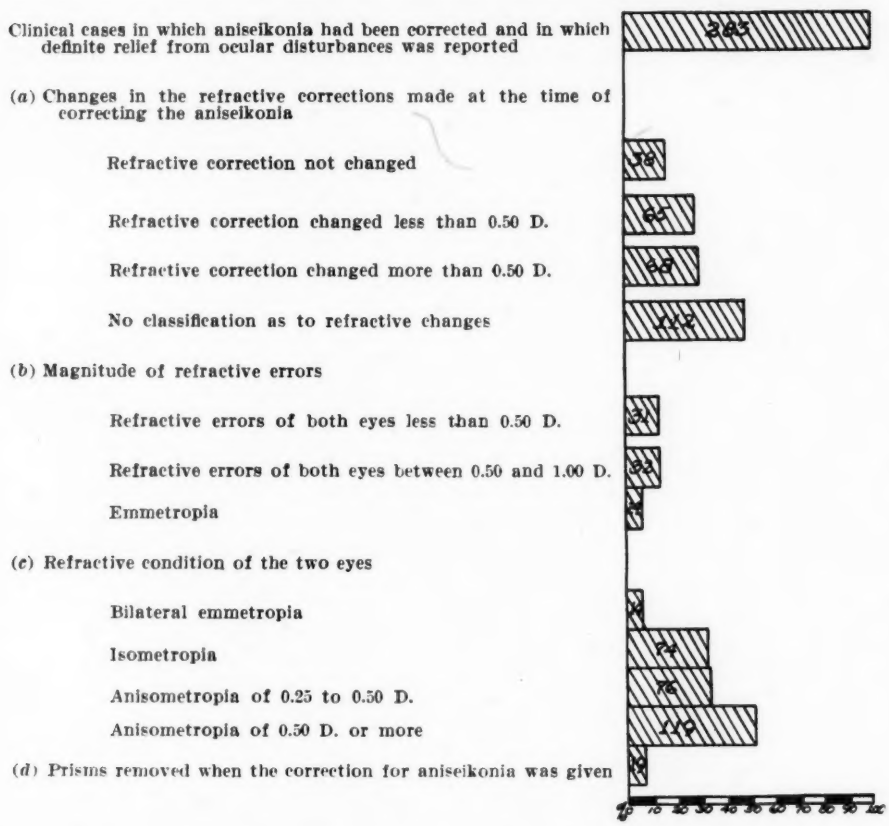
were examined for aniseikonia. Of this number, 625 were given glasses to correct their aniseikonia, and, so far as is known, 500 of these are now wearing such corrections. Between November 1934 and May 1936, reports were received from 425 patients to whom letters had been sent requesting information as to the relief obtained from the correction for their aniseikonia. Two hundred and eighty-three patients reported definite relief from the particular symptoms of which they had complained. This number of cases in which the aniseikonia had been corrected successfully seemed to be sufficient to warrant a practical statistical study of the relationships between aniseikonia and ametropia.

STATISTICAL RESULTS

The tabulation of the responses to inquiries sent to the 500 patients who are wearing corrections for aniseikonia is given in the table and

shows that 283, or 57 per cent, have obtained definite relief from certain symptoms of ocular discomfort. These figures agree closely with those found at other clinics [3] where aniseikonia is measured and corrected.

TABLE 2.—*Graphic Representation of the Data of Table 1* *



* The number of cases is given opposite each classification, and the corresponding percentage value can be read by means of the scale at the bottom of the figure.

COMMENT

It has been suggested that the relief obtained by persons with aniseikonia may have been due to a change in the correction for ametropia. Data of the table and figure show that for 38 of the 283 patients successfully treated no change in the correction for ametropia was made in the prescription which also corrected the aniseikonia. Since

3. Footnotes 1 *c*, *d*, *e* and *f*.

small refractive changes, of less than 0.5 D., are generally held to be insignificant, we may add to this group 65 other patients, for whom the change did not exceed 0.37 D. Thus, one finds 103 patients for whom no significant change in the correction for ametropia was made in the prescription which also corrected the aniseikonia. This combined group represents 36 per cent of the 283 patients who reported relief. We may, then, safely conclude that the relief from ocular discomfort obtained by these patients resulted, for the most part, from the correction of the aniseikonia.

There were, however, 68 patients for whom a radical change in the refractive correction seemed necessary or advisable. An attempt was made in many of the cases to determine whether relief could be obtained by means of the new refractive correction alone. In some of the cases the financial or physical condition of the patient did not permit such controlled experimentation.

The remaining 112 patients, or 40 per cent of the total number of 283, could not be classified in this manner for one of the following reasons: (1) The glasses had been discarded because no relief had been obtained; (2) neither glasses nor any record of previous prescriptions was available; (3) glasses had never been worn before.

At a recent conference a group of ophthalmologists expressed considerable interest in the degree of refractive error present in persons in whom aniseikonia has been demonstrated. Of the 283 patients who obtained relief by glasses for correction of aniseikonia, 63, or 22 per cent, were found to have a refractive error of 1 D. or less. This group may be subdivided as follows: 31 patients having a refractive error of 0.50 D. or less and 32 patients having a refractive error varying from 0.62 to 1.00 D.

While persons who have the condition true emmetropia are relatively rare, 11 patients with this condition have been examined and treated at The Eye Clinic. These patients, however, had definite symptoms of ocular discomfort, and although the cause of this ocular discomfort could not be found, they had been told repeatedly that the basis of their trouble was not ocular. For the reasons already stated, we are justified in adding to this group 3 patients whose refractive errors did not exceed 0.25 D. and who obtained complete relief by means of the correction for their aniseikonia without the use of a refractive correction. This gives a total of 14 patients, which represents 5 per cent of those reported on.

Just as there are persons with emmetropia who have aniseikonia, there are many more for whom the correction for ametropia is exactly the same for both eyes. Correction for aniseikonia has been included in the prescription for 74 patients with isometropia. This

fact shows that the correction of the refractive error in these cases is not the cause of the aniseikonia.

That aniseikonia is due to the correction of anisometropia is only partially true. Cases do occur in which, apparently, the aniseikonic magnification compensates for the aniseikonia produced by the correction for anisometropia. In the group of 283 patients under discussion, 195, or 69 per cent, were anisometropic; that is, there was a difference of refraction between the two eyes of 0.25 D. or more in one or both of the principal meridians. In order to clarify the relationship between anisometropia and aniseikonia a statistical treatment was made of the data for 119 patients who showed anisometropia of 0.50 D. or more in one or both of the principal meridians. In each case the refractive difference between the two eyes in each of the eikonic meridians (i. e., the meridian in which the aniseikonia was measured) was computed, and this difference was then compared with the difference in size actually measured in the clinic. Thus, the correction of astigmatism occupies an important place in such analysis. The results of the comparisons of the meridians may be summarized as follows:

1. The total number of meridians in which comparisons were made was 184.

2. The number of meridians in which the aniseikonia measured was in the same direction as that which might be expected from the correction of the anisometropia in those meridians was 136, or 74 per cent of the total number.

3. The number of meridians in which the aniseikonia measured was in the *opposite* direction from that which might be expected from the correction of the anisometropia in those meridians was 28, or 15 per cent of the total number.

4. The number of meridians in which either no aniseikonia was present, though anisometropia was corrected, or aniseikonia was measured, though no anisometropia existed, was 20, or 11 per cent of the total number.

This summary shows, first, that in the majority of the meridians, 74 per cent, the differences in size were in the direction that might be expected from the differences in power. This suggests that a part of the aniseikonia found had been introduced by the correction of the astigmatic ametropia. Second, in 15 per cent of the meridians the differences in size were in the direction opposite that which might be expected from the differences in power. This fact indicates that the basic aniseikonia was actually reduced by the correction of the astigmatic error. Finally, in 11 per cent of the meridians either aniseikonia existed without anisometropia or anisometropia existed without anisei-

konia. This result shows that the aniseikonia found was independent of the ametropic condition.

Further statistical analysis, by correlation of the data obtained in these 119 cases, gave a coefficient of correlation of 0.6[4] between the differences in size and the differences in power. Such a correlation shows that, although there is a general tendency for the correction of anisometropia to contribute to the amount of aniseikonia, no one to one relationship exists; that in certain cases of anisometropia there may be no aniseikonia at all, while in others the aniseikonia may be partially or totally reduced rather than increased by the correction for anisometropia. Furthermore, as has already been mentioned, since 74 patients with isometropia and 14 patients with bilateral emmetropia showed significant amounts of aniseikonia, and since there were 20 meridians in which there was anisometropia without aniseikonia, or vice versa, it is evident that aniseikonia may occur independently of the refractive error. It can be definitely stated, therefore, that on the basis of the ametropia present in any specific case no arbitrary deductions can be made as to the probable amount of aniseikonia.

Finally, more definite relief from ocular disturbance was obtained in 19 cases of heterophoria when the correction of aniseikonia was substituted for the correction of heterophoria by prisms. This fact shows that the heterophoria did not play a part in the etiology of the subjective disorders in these cases.

Five clinical cases have been selected which illustrate the various combinations of refractive conditions that may occur in cases of aniseikonia.

TYPICAL CASES

CASE 1.—Mr. H. G. R., a business man aged 53, was wearing the following correction on Dec. 10, 1929: a —3.75 D. sph. ⌒ + 4.25 D. cyl., ax. 10 ⌒ 1.00 D. prism base in for the right eye and a —3.00 D. sph. ⌒ + 4.00 D. cyl., ax. 165 ⌒ 1.00 D. prism base in for the left, with the addition of a + 1.75 D. sph. for each eye.

For six years the patient had suffered from almost constant headaches in the frontal and occipital regions, which were frequently accompanied by gastric symptoms. His eyes became excessively tired during near work, and he experienced pain and a burning sensation in the eyeballs, as well as photophobia. He was unable to do near work except for short intervals not exceeding a total of an hour a day. The patient was not robust, but no systemic conditions had been found that would account for his distress. His refractionist had been unable to relieve the trouble, and he was referred to The Eye Clinic.

The same refraction was found, and tests for heterophoria showed exophoria of 1 prism diopter for distance and exophoria of 6 prism diopters for near vision. The examination for aniseikonia showed that the right ocular image was

4. The prediction value of a coefficient of correlation of 0.6 is only 20 per cent.

smaller than the left by 1.75 per cent in the horizontal meridian. The correction for aniseikonia was given in a fit-over form, the original refractive correction without prisms being used. The patient returned five months later, on May 20, 1930, and reported that generally his eyes had given him less trouble than previously. There was still discomfort, but the periods of severe headaches were farther apart. He was reexamined in November 1930 and in April 1931. Although gradual improvement was reported at each visit, the basic prescription was not changed, but the correction for size was modified slightly.

In September 1934, after a careful recheck of the findings, the original refractive prescription was again given, together with a further, though slight, change in the correction for aniseikonia. The prescription was as follows: a —3.75 D. sph. ⌒ + 4.25 D. cyl., ax. 10 ⌒ 1.50 per cent meridional magnification, ax. 90 for the right eye and a —3.00 D. sph. ⌒ + 4.00 D. cyl., ax. 162 ⌒ 0.75 per cent meridional magnification, ax. 180 for the left. This correction was made up in permanent form as bifocal spectacles, with an addition of + 2.25 D. for each eye. In July 1935 he reported that these glasses were satisfactory and that it was possible for him to do more reading, since his symptoms had gradually disappeared.

CASE 2.—Mrs. A. W., a housewife aged 35, first visited the clinic on July 31, 1934. For the past fifteen to eighteen years she had suffered rather constantly from ocular fatigue, photophobia and car sickness and had occasional frontal headaches. The symptoms became much worse after a half-hour of reading or viewing motion pictures. Her ocular symptoms were accompanied by general physical fatigue and increase of nervous tension, for which repeated medical examinations had failed to disclose any organic basis.

Examination with the ophthalmo-eikonometer verified the emmetropic condition indicated by objective and subjective tests of refraction. There was esophoria of 0.25 prism diopter for distance and exophoria of 2 prism diopters for near vision. The left ocular image was found to be smaller than the right by 0.75 per cent in the vertical meridian and by 1.50 per cent in the horizontal meridian. The correction for this condition was given in a temporary prescription on July 31, 1934.

The patient reported partial relief after a short time, and as she continued to wear the glasses the symptoms of long standing gradually disappeared. In June 1935 she reported that she could read or do close work for from seven to eight hours a day without becoming nervous or tired and that the photophobia had completely disappeared. The car sickness had diminished so that the nausea had ceased, but long trips still caused general fatigue and a tired feeling around the right eye. On the basis of this report, glasses in a permanent form were furnished. In August 1935 she stated that her glasses continued to be a great source of comfort to her and that there was apparent improvement in her health.

At present, nearly two years after the first glasses for correction of the aniseikonia were given, she still has comfortable use of her eyes.

CASE 3.—Mrs. A. R., a housewife aged 63, first came to The Eye Clinic in April 1934 and complained of excessive lacrimation, tiring of the eyes during near work, and headaches in the frontal and occipital regions, which had been more or less constant throughout her whole life. When she read she became nauseated within a few minutes. When riding in a train or car she had to keep her eyes closed or refrain from looking out of the windows. She was afraid to cross streets because of extreme dizziness. Her physician had put her on a strict diet because of chronic biliousness.

On May 10 she was given the following prescription: a + 2.75 D. sph. ⊃ + 1.00 cyl., ax. 5 for the right eye and a + 4.75 D. sph. ⊃ + 1.75 D. cyl., ax. 5 ⊃ 1.25 per cent overall magnification for the left. One notes that the correction for the left eye is 2 D. stronger than that for the right and that the magnification added to the left lens for the correction of the aniseikonia is opposite to what one might expect. In a few weeks she reported marked improvement. She stated that she had not had any headaches, that she was not as nervous as formerly and could cross streets without fear and that she could read and sew without trouble. The car sickness and biliousness had disappeared, and her diet was not limited.

Two years later, in May 1936, she reported that she had fewer headaches than previously and these were rarely accompanied by nausea, that she could read comfortably and no longer rode with her eyes closed or feared to cross the street.

CASE 4.—Mrs. B., a weaver in a mill, aged 33, was referred to The Eye Clinic by an ophthalmologist because his refractive findings confirmed the ones of the practitioner who had examined her the year before and because the amount of error did not seem sufficient to explain the severe symptoms of which she complained. A general physical examination was found to give negative results.

Although her general health was fairly good, she had suffered from migraine headaches for sixteen years. These occurred weekly and lasted from one to three days. The attacks started with severe pain in the right eye, accompanied by lacrimation, fatigue and photophobia, followed by nausea and vomiting which made it necessary for her to go to bed until a day after the symptoms had subsided. Reading and motion pictures tired the eyes and usually brought on a headache. Riding in trains or cars always brought on a severe attack. She was unable to do steady work, as she was able to be up only two or three days a week. With reference to the family history, it was found that her mother had had similar headaches for many years.

External examination of the eyes and examination of the fundi gave negative results. The following refraction was obtained: with a —0.75 D. sph. ⊃ —1.50 D. cyl., ax. 90 vision of the right eye was 6/10, and with a 0.00 D. sph. ⊃ —0.25 D. cyl., ax. 180 vision of the left eye was 6/6. There were a fairly normal muscle balance for 20 feet (6 meters), exophoria of 2 prism diopters for this distance and exophoria of 7 prism diopters for near vision.

Examination with the ophthalmo-eikonometer in December 1934 revealed a moderately marked difference in size in the images of the eyes, which was in such a direction and of such an amount as to equal the aniseikonia produced by the correction for the anisometropia. A prescription was given which incorporated with the refractive correction already given a magnification of the right image of 2 per cent in the horizontal meridian.

The patient wore these glasses for a month, during which time there was no recurrence of the attacks of migraine. Her eyes were more comfortable, and she experienced no visual difficulties when viewing motion pictures.

On May 31, 1935, she reported that her eyes were fine and that she was feeling much better generally. She drove 300 miles (483 kilometers) and said that it was the first time in years that she had been able to take an automobile trip without experiencing a "sick headache." She has had no recurrence of the migraine, and her eyes do not feel tired at night after a day's work in the mills as they did previously. During the past year she has returned to The Eye Clinic on three occasions to have the glasses adjusted, and each time she has reported that there has been no recurrence of the so-called migraine.

CASE 5.—Mrs. S., a housewife aged 63, was first seen on Nov. 9, 1933. Her chief complaint was ocular fatigue. She stated that her eyes became tired during near work, and that such work was followed by headaches in the occipital region and vertigo, which were diagnosed by her internist as a mild form of migraine. She had to limit her reading to periods of fifteen minutes.

Ophthalmoscopic examination failed to reveal any pathologic findings except a small atrophic spot at the inner margin of the disk of the left eye. Her refraction was as follows: a + 2.00 D. sph. ⌒ + 0.50 D. cyl., ax. 115 for the right eye and a + 2.50 D. sph. for the left, with the addition of a + 2.50 D. sph. for near vision. With this correction she exhibited esophoria of 16 prism diopters for distant vision and esophoria of 6 prism diopters for near vision. She was given a temporary prescription, which incorporated a lens for the correction of the aniseikonia before the right eye with a prism of 2 D. base out before both eyes. This arrangement did not prove to be satisfactory; so the lens for the correction of the aniseikonia was discarded and the prism power increased to 5 D. before each eye. The latter combination was worn from May to Dec. 8, 1934.

The patient reported that there was considerable relief and that she was able to work from four to five hours a day, although she still complained of an ache in the right eye and in the occipital region.

At this time she was seen by Dr. Bielschowsky. His examination revealed esophoria of 13 arc degrees with the eyes in the primary position, which remained the same on dextroversion; in that position it was combined with negative vertical divergence of 2 arc degrees, but it decreased slightly on levoversion and still more on supraversion and infraversion. For near vision he found esophoria of from 6 to 7 arc degrees.

It was decided to remove the prisms and to try the effect of only lenses for the correction of the aniseikonia for near vision. The prescription was made up as follows: a + 4.50 D. sph. ⌒ + 0.50 D. cyl., ax. 90 ⌒ 1.50 per cent meridional magnification, ax. 180 for the right eye and a + 4.00 D. sph. ⌒ + 0.50 cyl., ax. 90 ⌒ 1.75 per cent meridional magnification, ax. 110 for the left.

These glasses were used for all close work, and on Feb. 1, 1935, the patient reported: "These last lenses [for correction of the aniseikonia] are much more efficient than the preceding ones [prisms base out], and I do not see why they should not be called very successful. I did not expect a pair of new eyes, and I am far more comfortable than I ever expected to be. I can work five or six hours a day on difficult and demanding work."

The patient was examined again by Dr. Bielschowsky on February 15; he found, with the eyes in the primary position, esophoria of from 12 to 13 degrees which remained nearly the same on dextroversion and levoversion and decreased on supraversion and infraversion. For near vision, with the refractive lenses alone, the patient exhibited esophoria of from 10 to 11 arc degrees, and with the lenses for the correction of the aniseikonia added, esophoria of only from 7 to 8 arc degrees.

A third examination was made by Dr. Bielschowsky on April 9. Esophoria of a similar variation was found, being of from 14 to 15 arc degrees for distant vision and of from 6 to 9 arc degrees for near vision.

This patient presented a condition of relatively high esophoria both for distant and for near vision, which remained practically the same throughout all the tests. Correction of the esophoria with only prisms base out brought partial relief, but correction of the aniseikonia *without recourse to prisms at all* brought full and permanent relief.

SUMMARY

The data presented in this report may be summarized as follows:

Of the 829 patients examined for aniseikonia at The Eye Clinic, 625 were given prescriptions for the correction of their aniseikonia, and 500 of these were wearing the corrections at the time this study was begun.

Of the 500 patients to whom questionnaires were sent, 425 replied relative to the efficacy of the glasses for the correction of their aniseikonia.

Two hundred and eighty-three patients reported definite relief from ocular and general symptoms following the correction of their aniseikonia.

For 103, or about one third, of these patients no significant change was made in the refractive condition; hence it may be concluded that the relief obtained was a result of the correction of the aniseikonia.

For 68 patients, or about one fourth of the entire number, a significant change was made in the refraction, which leaves equivocal the results obtained in this group.

The cases of the 14 patients with emmetropia and the 74 with isometropia in whom significant amounts of aniseikonia were corrected show that aniseikonia may exist *independently* of ametropia.

Since 195, or 69 per cent, of the patients with aniseikonia were anisometropic, the presence of anisometropia suggests the possibility of concomitant aniseikonia.

The coefficient of correlation of 0.6 between aniseikonia and anisometropia indicates that, although a correction for ametropia may be a partial cause of aniseikonia in the general run of patients, there are also many persons in whom either there is no relation between the two or the correction of the ametropia may tend to reduce rather than to increase the basic aniseikonia.

The fact that the patients with emmetropia and isometropia showed aniseikonia of a sufficient amount, together with the fact that a coefficient of correlation of 0.6 was found between aniseikonia and anisometropia, show that on the basis of the ametropia no arbitrary deductions as to the possible amount of aniseikonia which may be present in any specific case are allowable.

When the correction for the aniseikonia was substituted for the prisms which had corrected marked heterophoria in 19 cases, definite relief from symptoms resulted.

CONCLUSIONS

The clinical results so far obtained justify the following conclusions: First, one of the important steps in obtaining efficient binocular vision is the correction of aniseikonia; second, in the cases of all patients

with functional disturbances of the eyes, including those who have failed to obtain relief by the correction of their refractive errors or motor anomalies, a clinical investigation for aniseikonia should be made; third, on the basis of the ametropia presented by any specific patient no arbitrary deductions as to the probable amount of aniseikonia can be made, and, finally, since anisometropia and aniseikonia are often concomitant, although not commensurate (or highly correlated), all patients with anisometropia should be examined for aniseikonia.

Prof K. N. Ogle and Mr. H. A. Imus assisted in the statistical analysis of the data and in the preparation of the manuscript.

LIGHT ADAPTATION AT THE MACULA

AN EXAMPLE OF ITS INDUSTRIAL IMPORTANCE

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PHILADELPHIA

This paper is the result of work done in the laboratory for ophthalmology at the Wills Hospital for the National Brotherhood of Railway Firemen and Enginemen. The study was made in connection with the petition of this body before the Interstate Commerce Commission to compel the installation of automatic stokers on all coal-burning locomotives. This petition applied to all railroad runs whereon it is necessary for the firemen to be on the lookout for track signals, and to all occasions when they (the firemen) are specifically charged with the correct calling of such signals, as to both noting them on time and calling them correctly.

There were some other important factors connected with this petition before the Interstate Commerce Commission, such as economy of operation and the usual fatigue connected with firing by hand, but this paper has to do only with the factor just mentioned.

Firemen claimed before the commission that the effects on their eyes of the illumination from the fire box made it impossible for them to see the signals and to call them properly. This claim was based on their practical experiences and had only one important and serious point; i. e., after firing they were blinded for a certain length of time, during which it was impossible for them to see and to call the illuminated railroad signals.

Considering the speed of a railroad train when it is traveling with the throttle wide open and the distance which it covers in the time during which they were temporarily blinded, the firemen were aware of the dangers connected with such unguarded running. Disciplinary measures were being applied so frequently to the firemen because of their apparent disregard of signals, discovered when checking their observations against the observations of the driver of the locomotive, that they felt they were compelled to petition the Interstate Commerce Commission as they did.

From the Laboratory for Special Examinations, the Wills Hospital.

Read before the Section of Ophthalmology of the Philadelphia College of Physicians, March 19, 1937.

In the final hearing various methods of protection, such as filters, goggles and visors, were discussed, but each had definite and serious defects. In the final analysis, protection from the illumination was the cause, and any protection from the illumination hindered the railroad man in his subsequent lookout for signals.

The laboratory work which had to be done in connection with this problem was: (1) simulation of the illumination of a fire box, (2) adaptation of persons to this illumination, (3) the simulation of a three point signal light with its illumination corrected for various distances and (4) determination of the effects of the illumination of the fire box on an observer's reaction time for finding and for calling correctly the simulated three point position light signal. The illumination of this signal was computed and adjusted for distances of 100, 300, 500 and 1,000 yards (91, 273, 455 and 910 meters). Basic fire box illumination consisted of from 4,000 to 5,000 foot-candles per square inch (6.3 sq. cm.) of the surface exposed, which is the usual illumination of a Mogul type of locomotive fire box, at from 36 to 38 inches (91 to 96 cm.) from the frame of the door of the fire box. Actual measurements with a Weston photometer at 6 feet (1.8 meters) from the fire box gave a reading of 500 foot-candles per square centimeter. The actual foot-candle power of the standard railroad signal light at 1 foot (30 cm.) from the housing was 50 foot-candles per square centimeter. The illumination at the housing for colored signal lights, considered as the standard for the green light, was 36 foot-candles per square centimeter. The available distance in the laboratory for simulating the three point position light was 30 feet (9 meters) from the observer to the position light.

The distances at which a fireman was exposed to the illumination of the fire box were carefully estimated under working conditions on the railroad run; the minimum was found to be 38 inches (96 cm.) and the maximum 6 feet (1.8 meters). Further, in estimating working conditions it was necessary to determine the lengths of time during which a fireman was exposed to this illumination. The distances at which the firemen were exposed were measured on a locomotive with a meter rule and the lengths of exposure were determined with a stopwatch. Six series of reaction times (generally accepted by railroad men) resulted from this.

Series A consisted of five exposures of 3 seconds each, with a 3 second interval, that is, an equivalent of the time required to open the fire box, throw in coal, close the door and repeat this process five times in ½ minute. Series B consisted of an unbroken exposure of 5 seconds; series C, of an unbroken exposure of 10 seconds; series D, of an unbroken exposure of 15 seconds, and series E, of an unbroken exposure of 25 seconds.

The test conditions in series B, C, D and E were connected with firing and with the trimming of the fire bed.

The test in series F consisted of ten exposures of 3 seconds each, with 3 second intervals between exposures. This was also connected with dressing the fire bed for obtaining maximum fire efficiency.

It was manifestly unnecessary to work with 500 foot-candles per square centimeter at 6 feet. A rheostatically controlled light was therefore used for the light adaptation, the illumination being reduced by the square of the working distance to a working distance 3 or 4 inches (7.5 or 10 cm.) from the eye. The foot-candle power of the position lights was similarly rheostatically controlled and was also reduced by the square of the distance to the proper foot-candle power, even to the third or fourth decimal place, depending on the distance simulated. The advantage of light adaptation at 8 to 10 cm. from the eye with a proportionately decreased illumination over utilization of a proportionately increased illumination at the actual working distance of 36 to 38 cm. from the eye is evident.

Under circumstances of dark adaptation the retina is normally able to perceive light in decreasing degrees of illumination even to an arbitrarily chosen normal of 0.00001 lambert. In light adaptation the reverse is true. Practically speaking, when the macula has been adapted or subjected to an illumination of a greater degree, exhaustion of the macular biochemical elements makes it impossible for the person to see lesser degrees of illumination until the effects of the greater illumination have disappeared. The macula, while sensitive so far as direct vision is concerned, becomes exhausted rapidly under increasing degrees of exposure to light. The sensitivity of the extramacular region increases by over one thousand times more than the macula itself when any problem in difference in light is present; but the fireman must use the macula for correct identification even though the signal was first perceived by an extramacular region.

The observers used in this study were firemen employed by the railroad. All were tested for visual acuity, for normal fields of vision and for a normal threshold for light after dark adaptation. The fundi were examined in each instance, and no one was used as an observer who did not have normal visual acuity and a normal fundus. Repeated readings were taken until each subject gave results which were consistent for that person. Subjects who gave high readings constantly (there were several such persons) were excused, and their readings were not utilized in the final recapitulation. The averages reported were the lowest recorded. Repeated tests were made on the subjects

finally selected, and their readings were presented in the hearing before the Interstate Commerce Commission. Table 1 gives the averages for the series:

TABLE 1.—*Averages of Reaction Time for the Series*

Series	Average Time, Seconds	No. of Observers
A	26	10
B	24	10
C	25	9
D	38	12
E	48	10
F	56	13

Though not included in the report of the findings, readings were found for series A, C and F as high as 2 minutes; for series E up to 9 minutes, and for series F and D of 2 minutes and 10 seconds and of 2 minutes and 40 seconds, respectively. The higher readings were not as frequent as the averaged lowest readings, but they appeared with disconcerting frequency. They are given in table 2.

TABLE 2.—*Averages of High Reaction Time for the Series*

Series	Average Time, Seconds	No. of Observers
A	60	3
B	68	3
C	64	4
D	90	1
E	98	3
F	102	1

All the observers were first subjected to dark adaptation to determine the presence of a normal curve for dark adaptation. Each observer was then taken separately and subjected to the light adaptation of the series under investigation. The light in the tests of light adaptation was controlled by a switch, and the duration of exposure was controlled with a stop-watch. The position light was placed 30 feet from the observer at varying degrees from his left side and at varying heights to simulate the actual working conditions. It was placed in the position of stop, go or caution. As soon as the light was switched off, the interval of time until the subject saw the position light and called it correctly was noted. His efficiency in perceiving colored lights was then tested. Tests with colored lights showed a delay in the perception of red light over yellow-white light of a minimum of 10 per cent; in the case of green light there was a decrease of 20 per cent, and in the case of blue light, a decrease of 100 per cent. It is interesting that this proved the usual considered theory in regard to the transmissibility of colors that when the relative efficiency in red, green and blue illumination is considered, matters are definitely in favor of red. Rice, Uhthoff and others have long held that the degree

of visual efficiency as regards visual acuity is highest in the perception of white light, next highest in the perception of red light, next highest in that of green light and least in that of blue light. For instance, in series E, in calling the red light the least interval was 1 minute and 10 seconds, while the greatest was 5 minutes. For the green light the least interval was 21 seconds and the greatest 5 minutes and 42 seconds. For the blue light the least interval was over 2 minutes and the greatest 7 minutes. It must not be forgotten that the subjects were all experienced railroaders.

Some interesting conclusions can be drawn from these findings. Their accuracy is incontestable. The physical set-up of the test was checked and rechecked, and if any errors were made they were in favor, constantly, of higher readings. This was proved by readings made in the cabs of locomotives; in almost every instance the intervals of time there determined when checked against the intervals determined in the laboratory were longer than the latter.

The effect of both night driving and day driving was included in the study. The findings were essentially the same in daylight driving as in night driving, undoubtedly because of the great degree of fire box illumination. Also, though nonilluminated semiphore signals replace, in part, the illuminated position lights, the central scotoma is present regardless of whether the signal viewed is a day-time semiphore or a night-time position light. Repeatedly the subjects described their finding of the approximate position of the signal light by their indirect vision and its subsequent disappearance within the field of the scotoma when they directed their macular vision thereon. When they were under the effects of the longer exposures a greater number of mistakes occurred in calling the signals, *after the signal light was seen.*

The increasing curve for the interval in series B, C, D and E is interesting. Five times the constant exposure did not give a proportionately longer interval. The interval is actually only twice as long. On the other hand, the ratio which exists between series A and series F is upset inversely. In series A the five exposures to light of 3 seconds each totaled 15 seconds of exposure and 15 seconds of darkness, while in series F there was 30 seconds of exposure with intervals of 30 seconds of darkness between the exposures. In series A the average was a 26 second interval, while in series F, in which the test condition was a condition frequently met in firing, the interval was slightly greater than twice that amount. It is interesting to compare these effects of light adaptation on visual acuity and on speed of discrimination and the disproportionate effects of increasing the illumination with certain established facts in regard to the variation of intensity of illumination. The summation curve is not a regular one; instead it constantly

approaches or tends to approach a vertical line of maximum efficiency, regardless of increasing illumination.

For instance, Ferree and Rand showed that in using illuminations ranging from 0.001 to 20 foot-candles there was a gain in visual acuity, with an increase from 0.001 to 0.01 foot-candle, or 489 per cent. With an increase from 0.01 to 1 foot-candle there was a gain of 63.7 per cent. With an increase from 1 to 5 foot-candles a gain of 30 per cent occurred, and with an increase from 5 to 25 foot-candles a gain of only 8.2 per cent occurred. Rice's figures were somewhat differently expressed but illustrate the same point. According to his findings, with an intensity of 0.015 foot-candle visual acuity was 0.386. With an intensity of one hundred times this amount, that is, 1.5 foot-candles, there was visual acuity of 1.464, while with an intensity of one thousand times the amount visual acuity was increased to only 2.087. With three times the last amount, or three thousand times more than the original intensity, visual acuity was increased to only 2.2—a total gain over the original efficiency of but 17.54 per cent. In regard to the effect of the intensity of light on the speed of discrimination, with an illumination of 0.4 foot-candle, according to Ferree and Rand, the speed of discrimination was 3.525 seconds. With an illumination of ten times this amount, or 4 foot-candles, the speed of discrimination was 8.654 seconds. With an illumination of forty-eight times this amount the speed of discrimination was 23.81 seconds, which is less than eight times the speed obtained with the lower degrees of illumination. In regard to the effect of the intensity of the light on the speed of adjusting the eye for clear seeing at different distances, with an illumination of 2.04 foot-candles there was a normal reading of 0.411 second. With an illumination five times this, that is, 2 foot-candles, the reading was 0.568 second, while with 12 foot-candles, that is, an illumination forty-eight times the amount of the original, there was a further increase of only 0.316 second. These findings are quoted only to show the effects which varying degrees of illumination have on the macular region in that increasing the illumination does not increase the efficiency of discrimination proportionately. They are concurred in not in actuality (for confirmation of these findings was not the problem of our laboratory) but decidedly in substance, for we discovered similar relationships with the very high and the very low degrees of illumination.

The findings were reported before the Interstate Commerce Commission, and testimony was given thereon. The decision of the Interstate Commerce Commission is not especially relevant to the presentation of this work. The work is presented simply as an application of laboratory methods in an attempt to solve a serious industrial problem which can have a far reaching effect both in safeguarding the lives of railroad

passengers and in making necessary or unnecessary the expenditure of huge sums of money by the railroads for equipment not considered by some a definite necessity.

1930 Chestnut Street.

DISCUSSION

DR. FRANCIS HEED ADLER: I should like to call attention to the fact that each determination was made immediately after exposure to the artificial fire box illumination. It would not be likely that under actual conditions the signals would come each time after the fireman had looked into the fire box. Unless one had data showing how often signals are presented and how often the fire box must be opened one could not tell what the chances were of the moment after exposure to fire box illumination and that at which signals come coinciding, and since the scotoma lasts a relatively short time I do not believe that this is the main reason why so many signals are missed.

DR. H. MAXWELL LANGDON: Dr. Spaeth said one thing that I thought was a slight exaggeration: that a train may run uncontrolled for a short time. Is that correct?

DR. EDMUND B. SPAETH: The average firing conditions on the short runs between Philadelphia and Atlantic City, for instance, were determined. On the longer runs, where there are miles without signal lights, these conditions, of course, would not apply. In answer to Dr. Langdon: My statement implying that a train may run uncontrolled for a short time may be a slight exaggeration, and the explanation should be made that there are many times, as stated at the final hearing, when signals are visible only to the fireman and not to the engineer.

ASTROCYTOMA (TRUE GLIOMA) OF THE RETINA

REPORT OF A CASE

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The purpose of this paper is to report an apparently unique retinal tumor. So far as can be found, no similar tumor has been reported in the literature. This tumor is a true glioma of the retina—an astrocytoma.

The term glioma of the retina was first used by Virchow to describe tumors now known as retinoblastoma. It was later pointed out that such tumors were not glial in nature and differed essentially from the true gliomas of the brain to which they had been likened. Wintersteiner,[1] following Flexner's[2] lead, introduced the term neuro-epithelioma retinae, and Verhoeff [3] suggested the better name retinoblastoma, by which the tumor is now known in this country. With the exception of a few French authors,[4] ophthalmologists have abandoned the term glioma of the retina, generally believing such an entity does not exist. However Verhoeff in 1932,[5] in reviewing the subject of glioma of the optic nerve, called attention to the fact that there was true glial tissue in the retina and that sooner or later a true glioma of the retina might occur. The finding of the tumor reported here bears out this prophecy.

Glial tissue has been noted before in primary retinal tumors, having occasionally been demonstrated in the rare tumor of the pars ciliaris retinae first designated as teratoneuroma [6] and later renamed diktyoma.

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.

1. Wintersteiner, H.: Das neuroëpithelioma Retinae, Vienna, Franz Deuticke, 1897.

2. Flexner, S.: A Peculiar Glioma (Neuroepithelioma?) of the Retina, Bull. Johns Hopkins Hosp. **2**:115, 1891.

3. Verhoeff, F. H.: Tr. Am. Ophth. Soc. **24**:38, 1926.

4. Morax, V.: Pathologie oculaire, Paris, Félix Alcan, 1929. Redslob, E.: L'histogénèse des tumeurs de la rétine, Bull. Soc. d'opht. de Paris, March 1934, p. 123.

5. Verhoeff, F. H.: Tumors of the Optic Nerve, in Penfield, Wilder: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1027.

6. Verhoeff, F. H.: A Rare Tumor Arising from the Pars Ciliaris Retinae (Terato-Neuroma), Tr. Am. Ophth. Soc. **10**:351, 1904.

7. Fuchs, E.: Wucherungen und Geschwülste des Ciliarepithels, Arch. f. Ophth. **68**:534, 1908.

In these instances it has been present as a supporting framework and not as a part of the primary tumor. More recently a similar glial network has been noted by Grinker[8] and illustrated in detail in an article by Urra[9] as it appeared in the much more common retinoblastoma. In these tumors also the glial framework, like the nutrient vascular supply, is strictly a secondary proliferation and independent of the true tumor.

Von Hippel's disease and Lindau's disease[10] were once believed to be closely related to primary gliomatous growths in the retina. However, Lindau [11] recently has shown this is a misconception, these tumors being true angioblastomas with only slight and secondary glial proliferation.

Van der Hoeve[12] has described a peculiar cystic tumor sometimes found in the fundus of patients with tuberous sclerosis. The growth is typically cystic, edematous and degenerative.[13] Histologic evidence of low grade inflammation is common, while ruptures of the cysts, local implantation metastases to distant areas of the retina and free cysts in the vitreous seem to be the rule rather than the exception. The site of origin is usually, if not always, in the nerve head. In the histopathologic description of such a tumor[14] are mentioned areas with mere swelling of the elements of the nerve fiber layer and similar areas where there are also large cells said to be undifferentiated neurocytes. All the patients with a retinal lesion of this type also had typical tuberous sclerosis of the central nervous system, usually accompanied by the equally typical adenoma sebaceum and often by the less common visceral lesions.[15] There are certain points of similarity between the tumor reported here and this lesion, but there are also sufficient differences to clearly differentiate it from the phakomatosis just mentioned.

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8. Grinker, R. R.: Tumors of the Retina, in Penfield, Wilder: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1041.

9. Urra, F.: Ueber die feine Gewebsstruktur des Glioms der Netzhaut, Arch. f. Ophth. **112**:133, 1923.

10. (*a*) Meller, J.: Ueber das Wesen der sogenannten Hippelschen Netzhauterkrankung, Arch. f. Ophth. **85**:255, 1913. (*b*) von Hippel, E.: Ueber diffuse Gliose der Netzhaut und ihre Beziehungen zu der Angiomatosis retinae, ibid. **95**: 173, 1918.

11. Lindau, A.: Zur Frage der Angiomatosis retinae und ihre Hirnkomplikationen, Acta ophth. **4**:193, 1927.

12. van der Hoeve, J.: Eye Symptoms in Tuberose Sclerosis of the Brain, Tr. Ophth. Soc. U. Kingdom **40**:329, 1920; Augengeschwülste bei der tuberösen Hirnsklerose, Arch. f. Ophth. **105**:880, 1921.

13. van der Hoeve, J.: Eye Diseases in Tuberose Sclerosis of the Brain, Tr. Ophth. Soc. U. Kingdom **43**:534, 1923.

14. van der Hoeve, J.: Augenschwülste bei der tuberösen Hirnsklerose und verwandten Krankheiten, Arch. f. Ophth. **111**:1, 1923.

15. van der Hoeve, J.: Eye Symptoms in Phakomatosis, Tr. Ophth. Soc. U. Kingdom **52**:380, 1932.

Fig. 1.—Ophthalmoscopic appearance of the tumor.

UNIV OF MICH

The only other apparent glial tumefactions of the retina are the massive gliosis, described by von Hippel[10b] and by Friedenwald,[16] and the glial proliferation found either in massive, long-standing, inflammatory disease of the retina or as the end-result of degenerative processes.

The tumor reported in this paper is an isolated primary tumor of the retina which appears to be entirely distinct from all the aforementioned forms hitherto described.

REPORT OF CASE

History—Mrs. F. S., a 23 year old white woman, was first seen in the Wilmer Ophthalmological Institute on March 7, 1936. Two days before she had suddenly noticed that the vision of her left eye was very poor. There was no previous ocular history of any sort.

The family history was entirely irrelevant.

The past history was essentially unimportant, with no record of serious illnesses or injuries. There was a history of dysuria and frequency and urgency of urination of several months' duration. The only operation was a tonsillectomy at the age of 21.

General and Special Examinations.—The general physical and special neurologic examinations gave entirely negative results. Gynecological examination showed bilateral pyelitis, and cultures of urine obtained by urethral catheterization showed a moderate growth of Staphylococcus albus from each kidney and growth of Bacillus alkaligenes from the left kidney. Treatment was instituted later for this condition.

The blood counts, the blood chemistry and the cytologic picture were all normal. The Wassermann reaction of the blood was negative.

Examination of the Eye.—External examination showed both eyes to be normal, with normal pupils, tension and extra-ocular movements. Vision of the right eye was 20/15. Vision of the left eye was 20/70 and was not improved by refraction.

Ophthalmoscopically, the right eye showed clear media and a normal fundus. In the left eye the media were clear and the disk was normal. There was a mass in the left macular region, about 3 disk diameters in size, projecting forward about 8 D. and sharply circumscribed. It was smooth and whitish, with many vessels running over the surface. No extension could be made out, and the rest of the fundus was not abnormal in any way (fig. 1).

The visual field for form and that for color were normal in the right eye. The peripheral limits of the fields were normal in the left eye, but there was a large absolute scotoma extending nasally and somewhat down from the point of fixation for about 20 degrees.

The slit lamp examination gave entirely negative results for each eye.

A diagnosis of intra-ocular tumor in the left eye was made, and the eye was enucleated on March 9, with the patient under the influence of general anesthesia. The postoperative course was uneventful.

Pathologic Examination.—Macroscopic Observations: The globe was fixed in Bouin's solution and then opened in a frontal plant in the region of the ora

16. Friedenwald, J. S.: Massive Gliosis of the Retina, in Crisp, W. H., and Finnoff, W. C.: Contributions to Ophthalmic Science Dedicated to Edward Jackson, Menasha, Wis., George Banta Publishing Co., 1927, p. 23.

serrata. The anterior half was normal. In the posterior half neither the disk nor the vitreous appeared unusual. The retina and the choroid were in place. In the posterior pole, 4 or 5 mm. directly temporal from the disk, was a pale, almost white, smoothly rounded tumor about 5 mm. in diameter and 2 mm. high. No other lesions could be made out.

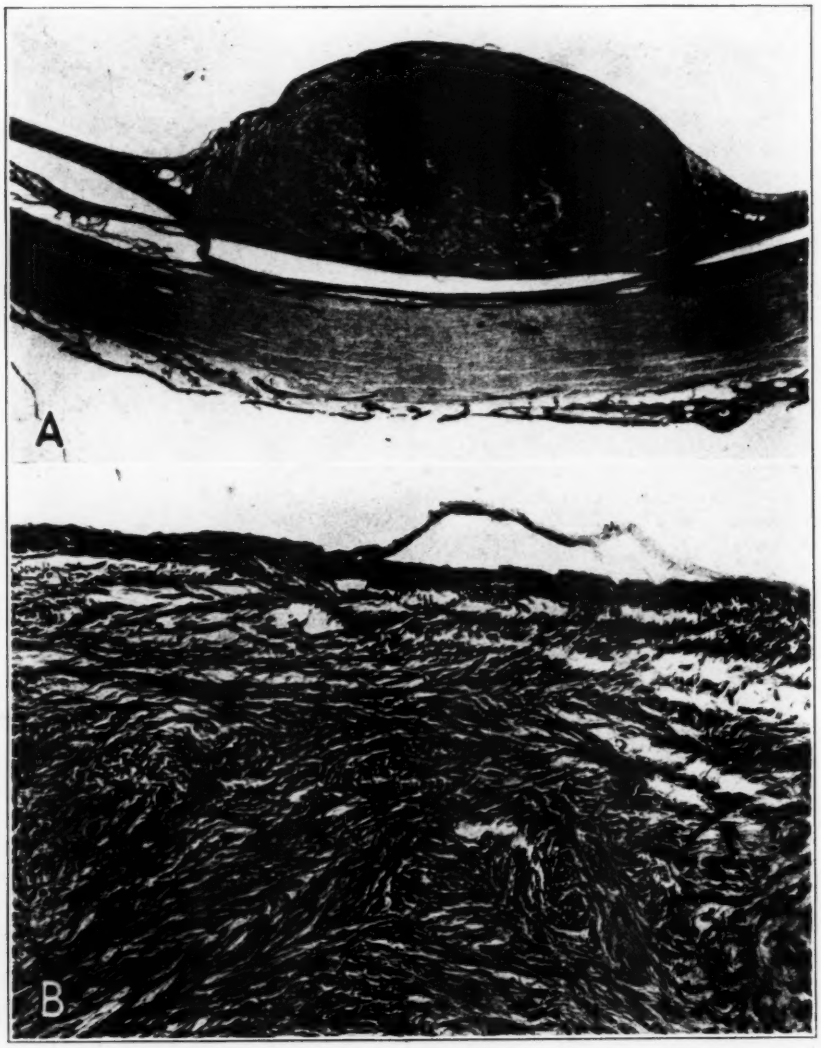

Fig. 2.—*A*, photomicrograph showing displacement of the normal retinal elements by tumor cells. *B*, photomicrograph showing the inner limiting membrane intact over the tumor.

Microscopic Observations: Horizontal sections were made and studied with the following stains: the hematoxylin and eosin stain, the Verhoeff-Van Gieson stain for elastic tissue, Mallory's aniline blue and phosphotungstic acid stains, the trichrome stain, silver impregnations and the Wilder stain for reticulum.

Except for the tumor the eye was entirely normal. The tumor was confined to the retina, supplanting and displacing the normal retinal elements (fig. 2*A*). The inner limiting membrane was intact over the tumor, which did not invade the

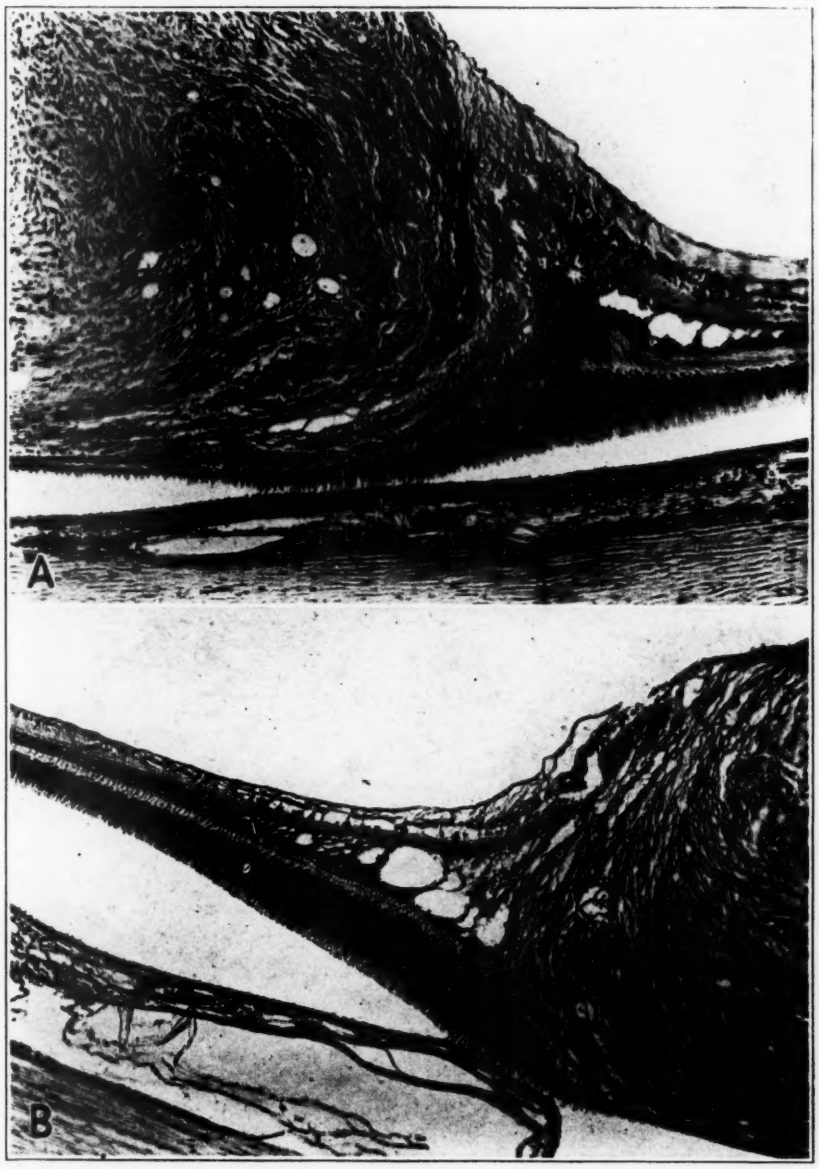

Fig. 3.—Photomicrographs showing lateral fading out of the tumor into normal retinal elements.

vitreous (fig. 2*B*). The rods and cones were for the most part preserved, but they were markedly thinned under the center of the tumor, where the pigment epithelium appeared compressed (fig. 4*A*). Bruch's membrane was intact, and

there was no invasion of the choroid. Laterally the tumor faded out into normal retinal elements (fig. 3). While there was a certain amount of either invasion or displacement of the outer layers, the tumor was predominantly in the inner layers of the retina and appeared to have arisen there.

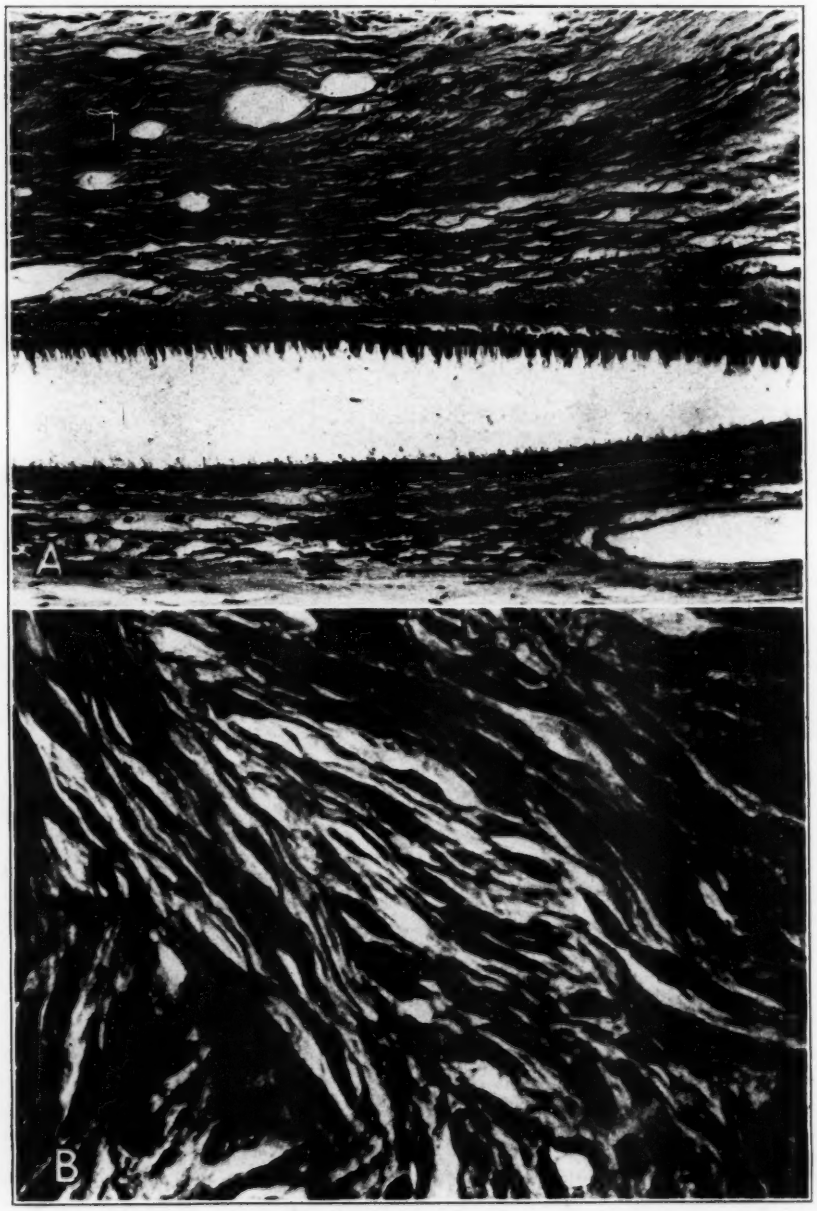

Fig. 4.—*A*, photomicrograph showing thinning out of the rods and cones under the center of the tumor, where the pigment epthelium appears compressed. *B*, photomicrograph showing long spindle cells with single, medium-sized, ovoid nuclei containing scattered masses of chromatin, a moderate amount of cytoplasm and long fibrillary processes.

The tumor was composed for the greater part of long spindle cells which had single medium-sized ovoid nuclei containing scattered masses of chromatin, a moderate amount of cytoplasm and long fibrillary processes (fig. 4*B*). Differential stains showed that these were neuroglial, not connective, tissue. There were also scattered clumps of large irregular cells with abundant eosinophilic cytoplasm and round or oval nuclei about twice the size of the nuclei of the ganglion cells in the normal retina. These stained fairly deeply, but many small granules and a few larger clear areas were made out within them. In many places the cytoplasm was drawn out into fibrillary axon-like processes. Occasional multinucleated cells were seen, and in some of the large cells the nucleus seemed to be distorted and displaced as if by swelling of the cytoplasm. Mitoses were excessively rare. There were many thin-walled blood vessels, but there was only a suggestion of astrocytic "sucker feet" about them. There was no communication with the choroid, and the blood supply seemed to be entirely from the retinal system.

COMMENT

Study of the cellular morphologic features of this tumor, together with its reaction to differential staining, leads to the conclusion that it must be classed as an astrocytoma arising from true gial tissue. The existence of fibrillary astrocytes in the inner layers of the retina has been recognized for years.[17] These are similar to the astrocytic glial elements of the central nervous system, where astrocytoma is the type of glioma most frequently encountered.[18] Perhaps the appearance of origin in the inner layers of the retina is significant in respect to the normal location of retinal astrocytes.

The age of the patient is of some interest. She was well beyond the usual age for retinoblastoma and in the age group for the remarkably rare retinal sarcoma[19] which originates in connective tissue of the retinal blood vessels. However, astrocytoma of the brain is usually found in the second and third decades of life[18]—in the age group of this patient.

The unusual retinal tumors in the phakomatosis of Bourneville (tuberous sclerosis) are the only lesions at all similar to the tumor in this case. That rare disease shows definite familial characteristics, whereas this patient's family history was irrelevant. Phakomatosis also involves the central nervous system primarily, with associated cutaneous and visceral lesions and occasional tumors of the eye. There was no evidence in this patient's history or in the results of the examinations

17. Greeff, R.: Die mikroskopische Anatomie der Netzhaut, in von Graefe, T., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1900, vol. 2, p. 74.

18. Bailey, P., and Cushing, H.: Tumors of the Glioma Group, Philadelphia, J. B. Lippincott Company, 1926.

19. Schick, F.: Das Perithelium der Netzhautzentralgefässe, Arch. f. Ophth. **81**:328, 1912. Elschnig, A.: Zur Kenntnis der primären Retinatumoren, ibid. **87**:370, 1914.

to suggest any such general or neurologic involvement. Vogt[20] reported a typical retinal nodule of tuberous sclerosis, the article being illustrated by an excellent colored drawing, and quite properly likened it to a mulberry. The ophthalmoscopic picture in the case reported by me contrasts with the ophthalmoscopic picture in Vogt's case. The cystic, degenerative, edematous character of the lesions in Vogt's case, together with their frequent rupture, reimplantation, and free nodules in the vitreous, further differentiate them from the tumor in the case I have just reported, in which all such features are conspicuously absent. Nevertheless, just as the microscopic picture in tuberous sclerosis of the brain bears some resemblance to astrocytoma of the brain, so a high power photomicrograph of one of the lesions in Vogt's case might show a picture resembling that seen in a high power photomicrograph of the retinal tumor reported here. This likeness cannot be entirely overlooked, but it seems impossible to group these conditions together in the face of so many points of differentiation. Perhaps with the possible accumulation of other cases of astrocytoma of the retina and more cases of tuberous sclerosis with associated retinal lesions a better understanding of the points of similarity and contrast may be had.

No definite opinion can be given on the relative malignancy of this tumor. Cytologically, it appears to be relatively benign. It is not particularly invasive. There are neither abundant mitoses nor other evidences of rapid growth. Metaplasia is not excessive. There is no information as to the time of onset of the growth, for the patient found the visual defect only accidentally, the other eye being normal. Since astrocytoma is the least malignant glioma of the brain,[18] by analogy this tumor should be of low malignancy. Since there are no evidences of metastases and enucleation has given thorough and complete removal of the growth, the prognosis should be good.

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20. Vogt, A.: Seltenen Maulbeertumor der Retina bei tuberösen Hirnsklerose, Ztschr. f. Augenh. **84**:18, 1934.

TREATMENT OF CAUSTIC BURNS OF THE EYE

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Chemical burns of the eye are of frequent occurrence. Cowan and Sinclair [1] in their recent review of blindness in Pennsylvania found that blindness was more often due to chemical burns than to detached retina. Detached retina has received much attention, while the study of chemical burns of the eye has been neglected.

In this paper I desire to discuss briefly the emergency treatment of burns of the eye caused by soluble acid and alkali and to present new experimental studies.

In advising treatment of caustic burns experimental work must be considered of paramount importance. The factors, such as (*a*) the time that has elapsed before initial treatment, (*b*) the amount of the caustic causing the burn, (*c*) the type of the caustic, (*d*) the strength of the caustic and (*e*) the type of treatment, are so variable that one cannot outline the proper treatment from clinical observation alone.

In a previous experimental study [2] on caustic burns it was found that, regardless of the concentration of a chemical or of the interval of time before treatment, the proper treatment was to irrigate the eye with water rather than to attempt neutralization. With neutralization chemical reaction with heat was apparent. Mechanical removal of the chemical was the most important factor. This was shown in the experiments with phenol, which is not soluble in water. The tremendous importance of the time that had elapsed before first aid was given was demonstrated. It was further shown that after a certain time of exposure for any concentration irreparable damage had been done. Some of the newer texts are advising treatment along the lines mentioned. A great many continue, however, to advise neutralization. The advocates of neutralization should have incontestable experimental proof of its desirability, as advising neutralization is practically the same as advising against any emergency treatment. Before a proper neutralizing solution could be obtained all the damage would have been done.

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1. Cowan, Alfred, and Sinclair, S. M.: Causes of Blindness in Pennsylvania from Medical and Social Aspects, J. A. M. A. **107**:757-760 (Sept. 5) 1936. Sinclair, S. M.: Personal communication to the author, Oct. 21, 1936.

2. Cosgrove, K. W., and Hubbard, W. B.: Acid and Alkali Burns of the Eye, Ann. Surg. **87**:89-94 (Jan.) 1928.

Fig. 1.—Normal rabbit eye. Note the transparent cornea, with the iris and the pupil clearly seen, and the normal lids. The nictitating membrane is prominent.

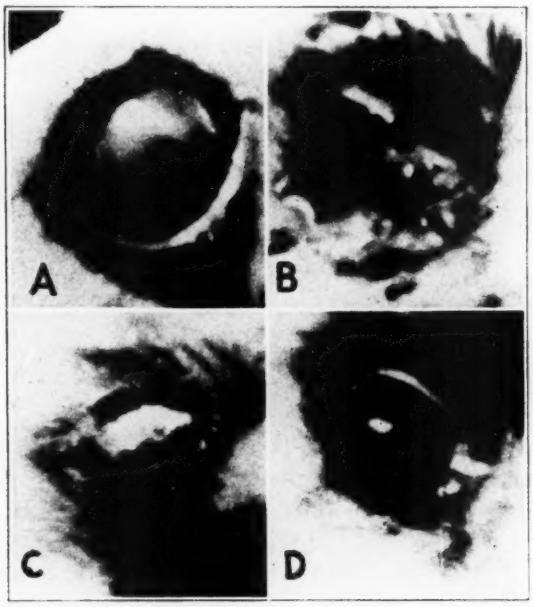

Fig. 2.—*A*, rabbit eye twenty-two days after exposure for thirty seconds to a 32 per cent solution of sulfuric acid. The eye was treated with vigorous irrigation with water. Note the rather clear cornea and the good condition of the eyeball. *B*, rabbit eye twenty-two days after exposure for thirty seconds to a 32 per cent solution of sulfuric acid. The eye was treated with vigorous irrigation with a 2 per cent solution of sodium bicarbonate. Note the partially degenerated eyeball. A comparison of *A* and *B* shows that treatment of an acid burn by irrigation with water is more efficacious than treatment by irrigation with a weak alkali. *C*, rabbit eye twenty-two days after exposure for ten seconds to a 20 per cent solution of sodium hydroxide. The eye was treated with vigorous irrigation with water. Note the completely degenerated eyeball. *D*, rabbit eye twenty-two days after exposure for ten seconds to a 20 per cent solution of sodium hydroxide. The eye was treated with vigorous irrigation with a 2 per cent solution of acetic acid. Note the almost normal eye. A comparison of *C* and *D* shows that treatment of an alkali burn by irrigation with weak acid is more efficacious than treatment by irrigation with water alone.

NEW EXPERIMENTAL STUDIES

In the experiments with caustic burns of the eye reported in this paper the effect of irrigation with water was compared with the effect of irrigation with a weak neutralizing fluid. With this procedure harsh chemical reaction was avoided. The mechanical factor of removal was present in both cases.

The effect was studied when a 32 per cent solution of sulfuric acid and a 20 per cent solution of sodium hydroxide were instilled into the eyes of rabbits. Eleven drops of caustic were used. The sulfuric acid was allowed to act for thirty seconds and the sodium hydroxide for ten seconds. These solutions finally destroyed useful vision in the majority of cases.

In these experiments I decided to irrigate the right eye with tap water in all cases. The left eye after the sulfuric acid burn was irrigated with a 2 per cent solution of sodium bicarbonate and after a sodium hydroxide burn with a 2 per cent solution of acetic acid.

Every possible precaution was taken to perform the experiments with accuracy. Two experienced assistants aided. The caustics and irrigating solution were carefully measured, and the time was regulated exactly by a stop watch.

Between 80 and 100 per cent of the acid burns treated by irrigation with a weak alkali were definitely worse than the acid burn of the opposite eye treated by irrigation with water alone.

Seventy-five per cent of the alkali burns treated by irrigation with a weak acid were definitely better than the alkali burn of the opposite eye treated by irrigation with water alone.

It is well known that acid proteinates are insoluble and that alkaline proteinates are soluble. The findings in these cases substantiate the theory that a soluble alkaline proteinate does not protect against further injury, although an insoluble acid proteinate does. These findings do not prove or disprove the value of irrigation, as all the eyes were irrigated in one way or another.

If sulfuric acid is a typical acid, an eye with an acid burn should be irrigated with water and not with a weak alkali. If sodium hydroxide is a typical alkali, an eye with an alkali burn should, when possible, be irrigated with a weak acid.

COMMENT

The findings in this group of eyes raise interesting points for additional experimental studies: 1. There are many more substances to be studied to determine the best emergency ocular treatment. 2. After-treatment of burns of the eye should be studied experimentally. Modified studies with tannic acid seem indicated. The present studies suggest that acidity of the conjunctival sac is advantageous after any type of burn. 3. The treatment of caustic burns of the skin and the gastro-intestinal tract should receive further attention experimentally. In

regard to the latter, it appears incorrect to prescribe a weak alkali for a person poisoned by an acid. This disagrees with treatment advised in the present literature.

SUMMARY

Acid burns of the eye should be treated immediately by irrigation with water and not with a neutralizing fluid—neutralizing substance in the water is harmful.

Alkali burns of the eye should be treated immediately by irrigation with a weakly acid solution. If this is not practicable, they should be irrigated with water and a weak acid should be instilled as soon as possible.

It is believed from these findings that further studies should be made of other parts of the body, such as the gastro-intestinal tract.

Further experimental studies of caustic burns of the eye are indicated.

PRIMARY CARCINOMA OF THE LACRIMAL SAC

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MINNEAPOLIS

Owing to the rarity of primary carcinoma of the lacrimal sac, the following case warrants recording.

REPORT OF A CASE

History.—E. J., a man aged 78, consulted me in June 1927 with regard to a mass over the right lacrimal sac. This growth was first noticed about two months prior to the time of the examination and had slowly increased in size. His only complaint was of epiphora.

Examination.—The vision of each eye was normal. Over the region of the right lacrimal sac was a firm, smooth, round mass approximately from 1 to 1.5 cm. in diameter. The skin was not red and was freely movable. Neither tears nor pus could be expressed from the puncta. A diagnosis of mucocele was made and removal advised.

Operation.—With the part under local anesthesia, the lacrimal sac was excised after the method of Meller. The anterior wall was much thickened and firmer than one would expect from an inflammatory condition. The sac contained a small amount of mucus. The wound healed per primam intentionem, and the patient returned home in three days. The appearance of the wall of the sac was suggestive of a new growth.

Microscopic Examination.—Dr. W. A. O'Brien, of the University of Minnesota, reported as follows: "The specimen was a small white tumor from eye. Sections showed nonpigmented cords and masses of epithelium resembling squamous epithelium. The diagnosis was carcinoma."

Second Operation and Course.—In August, eight weeks later, the patient returned, and a hard fibrous mass, 1.5 cm. in diameter, was removed by a second operation.

The patient was seen again in June 1929. There was a thickened elevated area 2.5 by 3 mm. on the inferior nasal portion of the cornea near the limbus. Two months later, in August, this area had spread to the limbus. This growth had involved the tissue beneath the conjunctiva, and there was slight limitation of motion of the eye. In May 1930 the patient was referred to Dr. Laura Lane for radium treatment. A pack containing 200 millicuries of radon was used at three distances, 1, 2 and 3 cm., with three different filters. The total dose amounted to 800 millicurie hours. On June 9, 600 millicurie hours was applied in a pack to the eyelid. When the patient was seen in March 1931 there was marked exophthalmos resulting from the extension of the growth backward. The cornea was cloudy and vision was reduced to counting fingers at 2 feet (61 cm.).

Third Operation.—In March 1931 exenteration of the right orbit was done, with the patient under nitrous oxide anesthesia. The eyelids were not removed. The postorbital space was found to be filled with a hard mass of tissue about 3 by 1 cm. in size.

Pathologic Report.—Dr. Charles Drake's report on the tissue was as follows: "Sections from the hard portion showed the structure to be made up of irregular masses of epithelium growing in all directions. Many mitotic figures were present. The microscopic diagnosis was carcinoma."

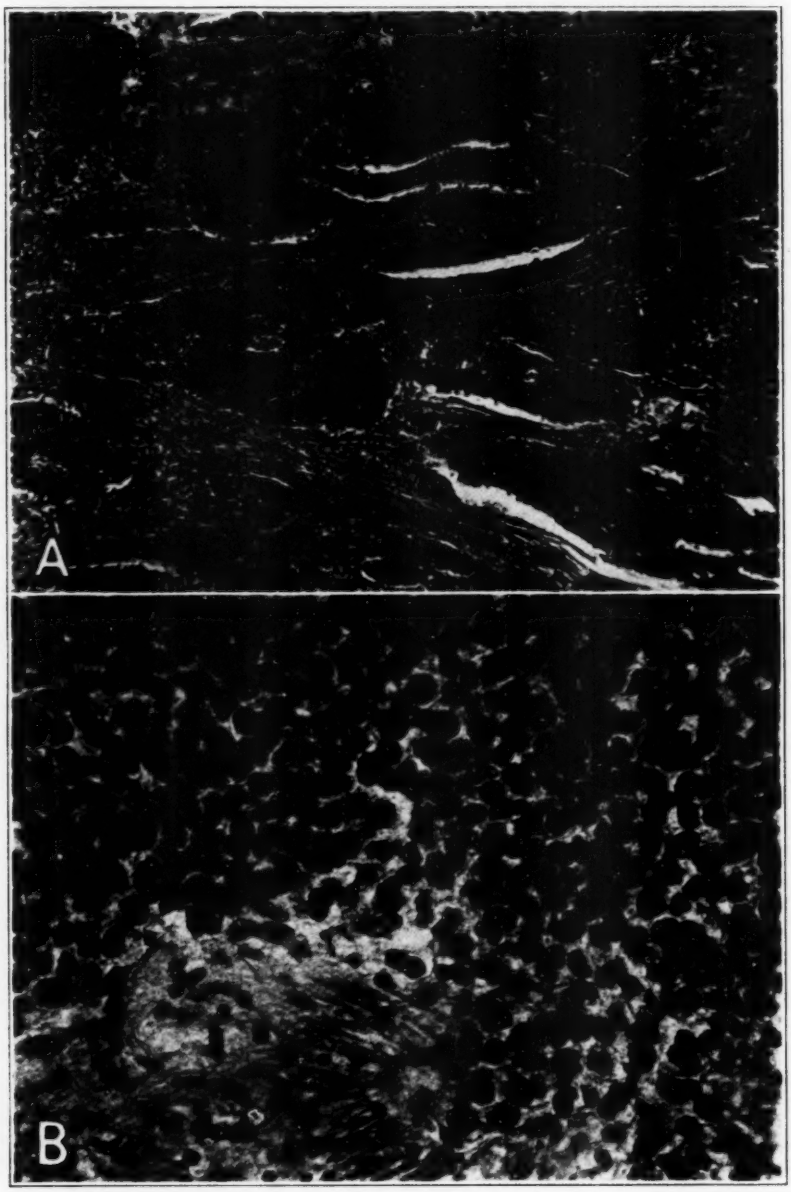

Fig. 1 (Spratt).—Carcinoma of the lacrimal sac. *A*, low magnification; *B*, high magnification.

Subsequent History.—The orbit filled in by granulation, and the lids became adherent to the scar tissue. The patient was seen again in April 1933, two years after the exenteration. There was no recurrence of the growth in the orbit.

There was, however, extensive metastasis to the submaxillary lymph nodes. He was referred to the University Hospitals for radium and roentgen treatment. Radon was implanted in the nodes on May 15 by Dr. Randall, who used a total of 28 millicuries in thirty-one gold implants. The thickness of the wall of gold was 0.3 mm. The total dose amounted to 3,690 millicurie hours. High voltage roentgen therapy was given to the right side of the face and neck over an area measuring 13 by 18 cm. on May 16 and 18. The factors were: 200 kilovolts, a 0.5 mm. copper filter plus a 4 mm. aluminum filter, a target-skin distance of 60 cm., 30 milliamperes, and a half value layer of 1 mm. of copper. The intensity used amounted to 33 roentgens per minute, and the total dose was 750 r. This therapy was followed by complete disappearance of the swelling. The patient died in July 1935 at the age of 86, eight years after the first appearance of the growth, four years after the exenteration of the orbit and two years after the radium treatment of the involved glands. There was no recurrence in the glands. The cause of death was coronary thrombosis.

Fig. 2 (Spratt).—Enlarged glands at the angle of the jaw. *A*, appearance of the patient two years after exenteration of the orbit; *B*, appearance two years after the implantation of radium and roentgen treatment and four years after exenteration of the orbit.

COMMENT

Synopsis of Cases.—Parsons [1] has described many types of tumor of the lacrimal gland but made no mention of a new growth of the lacrimal sac. Angiosarcoma, angiomyxosarcoma, cystoma, endothelioma, lymphoma, lymphosarcoma, fibrosarcoma, plasmasarcoma, malignant papilloma and carcinoma as primary tumors of the lacrimal sac have been reported.

A survey of the ophthalmic literature revealed sixteen additional cases of primary carcinoma of the lacrimal sac. In this group of seventeen cases, thirteen of the patients were males, and four were females. Thirteen were persons more than 40 years of age. One patient was aged 36; one was 25 years of age; one was a young woman of unknown age, and a fourth was a boy aged 18 (in this case the growth was a malignant papilloma).

1. Parsons, J. H.: Pathology of the Eye, New York, G. P. Putnam's Sons, 1904.

Synopsis of Seventeen Cases of Carcinoma of the Lacrimal Sac

Author	Year	Male	Female	Age, Years	Right	Left	Duration	Diagnosis	Recurrence	Cure	Unknown
Piccoli: Lavori d. clin. ocul. d. r. Univ. di Napoli **4**: 257-265, 1894-1896; abstr., Ann. di ottal. (supp.) **24**: 19, 1895-1896	1895	..	+	56	+		8 to 9 mo.	Carcinoma	In 3 to 4 mo.	.....	..
Sgrosso [13]	1900	+	..	65	Not given		4 yr.	Epithelioma	.....	Did not recur	..
Dalén: Beitr. z. Augenh., 1899, no. 41, pp. 1-9.....	1902	..	+	24		+	2 yr. after extirpation of sac	Carcinoma	In 4 mo.	.....	..
Lafon: J. de méd. de Bordeaux **36**: 172, 1906.....	1906	+	..	48		+	10 mo.	Epithelioma (cylindric cell)	Unknown	.....	+
Rollet [4]	1906	+	..	65	.....		.....	Epithelioma	Unknown	.....	+
Bistis: Arch. Ophth. **39**: 53, 1910.....	1909	+	..	60	Not given		.....	Carcinoma	Yes	.....	..
Pasetti: Ann. di ottal. **42**: 55-64, 1913.....	1913	+	..	73	.....		.....	Carcinoma	Not known	.....	+
Posey [7]	1921	+	..	74	.....		.....	Epithelioma (cylindric cell)	.....	In 2 yr.	..
Strada and Urrets Zavalia [11].....	1925	..	..	53	.....		2 mo.	Flat cell carcinoma	Patient died 10 mo.	.....	..
Bakker and Oudendal [12]	1926	..	+	Young woman	Not given		1 mo.	Basal cell carcinoma	+	In 18 mo.	..
Hilden [2]	1929	+	..	18	+		Several months	Malignant papilloma	In 3 mo. (2 operations)	In 2 yr.; 2 operations	..
Dupuy-Dutemps [3]	1929	..	..	47	+		4 to 5 mo. (papilloma removed at 22 yr. of age)	Papillary epithelioma	In 4 mo.	.....	..
Muirhead [5]	1933	+	..	60	+		5 mo.	Transitional cell carcinoma	Unknown	.....	+
Tennent [6]	1933	+	..	63	+		2 mo.	Cylindric cell carcinoma	Unknown	.....	+
Urrets Zavalia and Obregón Oliva [14].....	1935	+	..	36		+	1 yr.; epiphora	Epithelial origin	In 3 mo.	.....	..
Urrets Zavalia and Obregón Oliva.....	1936	+	..	47	.....		1 yr.	Carcinomatous	Gland at angle of jaw	In 4 mo.; 2 operations	..
Spratt	1936	+	..	78	+		2 mo.	Carcinoma	Orbit and angle of jaw; second operation performed	In 4 yr.; 3 operations	..

In two of these cases the growth was diagnosed as malignant papilloma (Hilden's [2] case and Dupuy-Dutemps' [3] case). In the latter case the patient had a papilloma of the lacrimal sac removed at the age of 22 years. When she was 47 years of age a malignant papilloma appeared at the site of the original tumor. This recurred after removal. (It is a question whether this case should be included in the series.)

The original articles were consulted when available, as titles frequently are misleading. As an example, the three cases of cancer described by Rollet [4] consist of one of primary carcinoma, one of sarcoma (polypus) and one of secondary melanotic sarcoma. Several writers have described carcinoma of the lacrimal sac secondary to tumor of the lid or antrum.

In the English literature, Muirhead [5] and Tennent [6] have each described a single case. The only case reported in the American literature is that described by Posey [7] in 1921.

De Vincentiis [8] (1876) is credited as having been the first to describe a primary cancer of the lacrimal sac. His original article was not available, but according to Silvestri,[9] who reviewed the Italian literature, the tumor was described as a papillary fibroma. Maggi [10] reported it as a tumor of the lacrimal sac.

The normal lining of the lacrimal sac is composed of cylindric cells. Repeated irritation due to chronic inflammation may cause this membrane to become thickened and form several layers of cells which change to the squamous variety. Consequently, microscopic examination may show the carcinoma to be the cylindric, the transitional or the squamous cell type.

Diagnosis.—Since the onset is insidious, with subjective symptoms no different from those of an ordinary cystic condition of the lacrimal

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2. Hilden, B.: Malignes Tranensackpapillom, zugleich ein Beispiel von Zellenmetaplasie in den Tranenwegen, Klin. Monatsbl. f. Augenh. **82**:661-665, 1929.

3. Dupuy-Dutemps, L.: Epithéliome papillaire de la fosse nasale consécutif à un papillome du sac lacrymal extirpé 24 ans auparavant, Bull. et mém. Soc. franç. d'opht. **42**:215-220, 1929.

4. Rollet: Trois cas de cancer non ulcéré du sac lacrimal, Arch. d'opht. **26**: 337, 1906.

5. Muirhead, W. N.: Carcinoma of the Lacrimal Sac, Tr. Ophth. Soc. U. Kingdom **53**:591-592, 1933.

6. Tennent, J. N.: Carcinoma of the Lacrymal Sac: Report of Two Cases, Tr. Ophth. Soc. U. Kingdom **53**:93-101, 1933.

7. Posey, W. C.: Report of a Case of Primary Tubular Epithelioma of Lacrimal Sac, Tr. Am. Ophth. Soc. **19**:205-208, 1921.

8. de Vincentiis, C.: Di un raro caso di fibroma papillare del sacco lagrimale, Movimento, Napoli **8**:418-423, 1876; quoted by Silvestri [9] and Maggi.[10]

9. Silvestri, A.: Sarcoma del sacco lacrimale, Ann. di ottal. **26**:452-459, 1897.

10. Maggi, F.: Contributo allo studio dei tumori primitivi del sacco lacrimale, Ann. di ottal. **35**:789-798, 1906.

sac, in only one of the cases in this series was the growth correctly diagnosed as malignant prior to operation. The appearance on examination is that of a hard, round mass with freely movable skin. Stenosis of the lacrimal duct is generally present.

In the first stage a correct diagnosis of carcinoma of the lacrimal sac is impossible, as epiphora is the only symptom. In the second stage, that of swelling, differentiation between a tumor and a mucocele can be made. A tumor gives rise to epiphora owing to pressure of the thickened wall of the lacrimal sac. Pressure on the sac causes little or no regurgitation, and the duct is patent on irrigation and on probing. Signs of inflammation are absent. In cases of mucocele the duct is generally obstructed. Pressure on a mucocele causes regurgitation of fluid into the nose or from the puncta on diminution in size of the swelling. A hard mass with rapid growth and lack of diminution in size on pressure indicates a new growth. In the third, or final, stage, characterized by rapid growth of the hard mass, ulceration and enlargement of the preauricular and the maxillary lymph glands, the growth presents little difficulty in diagnosis. The case reported by Strada and Urrets Zavalía [11] was the only one in which the growth was diagnosed as a malignant tumor before operation; in the other cases the growth was considered a mucocele. The only case in which pain was reported was that of Strada and Urrets Zavalía.

Prognosis.—These tumors are malignant, and recurrence is the rule. In five of the seventeen cases the end-results were not given. In three cases the growth was reported as cured. Posey's [7] case was observed two years; Bakker and Oudendal's [12] was observed eighteen months, and for the third, that of Sgrosso,[13] no time of observance was given. Recurrence was noted in nine of the seventeen cases. Two of the patients were operated on a second time; in one no recurrence was noted at the end of four months (Urrets Zavalía and Obregón Oliva [14]), and in the other, at the end of two years (Hilder [2]).

A third patient was operated on three times (my patient), and no recurrence was noted locally over a four year period. It is not unlikely that had these patients been traced, recurrence would have been found in all. There is no record of any patient having been free during the five year period.

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11. Strada, F., and Urrets Zavalía, A.: Contribución al estudio de las tumores malignos del saco lagrimal, Semana méd. **2**:1100-1108 (Oct. 29) 1925.

12. Bakker, C., and Oudendal, A. J. F.: Een geval van basalecullincarcinom van den ductus nasolacrimalis bij een inlandsche vrouw, Nederl. tijdschr. v. geneesk. **2**:2-6, 1926.

13. Sgrosso, P.: Epitelioma del sacco lagrimale, Ann. di ottal. **29**:82-96, 1900.

14. Urrets Zavalía, A., and Obregón Oliva, R.: Sobre un caso de carcinoma primitivo del saco lagrimal, Arch. de oftal. de Buenos Aires **10**:521-537, 1935.

Treatment.—All the patients were operated on (by extirpation of the sac with the tumor). Since the diagnosis of the malignant nature was made in only one case (that of Strada and Urrets Zavalía) prior to operation, removal was incomplete and recurrence was the rule.

After operation, Dupuy-Dutemps,[3] Strada and Urrets Zavalía,[11] Posey,[7] Urrets Zavalía and Obregón Oliva [14] (two cases), Muirhead [5] and I used radium or roentgen rays, with poor results. Local recurrence followed in all cases except those of Posey,[7] Strada and Urrets Zavalía [11] and mine. It appears that radium and high voltage roentgen therapy with proper filtration offer hope for cure if used after extirpation of a malignant tumor of the lacrimal sac.

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Clinical Notes

AN ENUCLEATION COMPRESSOR

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The instrument described in this paper is used during enucleation. Either the large or the small end is inserted into Tenon's capsule immediately after the optic nerve has been severed. By making pressure in

The enucleation orbital compressor.

the apex of the orbit one can compress the bleeding vessels of the orbital apex. This pressure will immediately stop the bleeding.

This instrument is held tightly against the apex of the orbit while the sutures are inserted in Tenon's capsule. The handle is made 10 inches (25.4 cm.) long in order that the assistant's hand holding the instrument may be out of the way of the operator. The shaft of the handle is small (6 mm. in diameter), so that it does not interfere while the operator is inserting sutures in Tenon's capsule.

The tips of this instrument are olive shaped; the large end measures 14 mm. and the small 11 mm. The large end is useful for operations on adults and children, and the small end is useful for operations on children under the age of 5 years.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

TUBERCULOUS LESIONS OF THE UVEAL TRACT

A REVIEW OF THE LITERATURE

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AND
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PHILADELPHIA

GENERAL CONSIDERATIONS

It is extremely difficult to prove that in a suspected case of uveal tuberculosis the condition is actually due to the tubercle bacillus. The history of the patient, the appearance of the lesion and the clinical course are all one has to rely on in making a diagnosis of uveal tuberculosis. A focal reaction to tuberculin is generally conceded, in this country, to be too dangerous to justify its use, and even when positive does not afford absolute proof of the nature of the lesion. The patient's cutaneous sensitivity to tuberculoprotein is a diagnostic aid, the significance of which we shall discuss later on. The absence of other possible etiologic factors to explain the lesion is further suggestive evidence. The Schilling differential blood count has been reported by Bredeck [1] as of diagnostic value, taken in conjunction with the subcutaneous tuberculin test. Other inflammatory conditions may also cause a similar shift in the blood picture, so that the test is not specific, and a diagnosis of tuberculosis cannot be made on the basis of the blood picture alone. Vernes' test has been used with some success by Williamson-Noble.[2] The test is sometimes positive in the absence of tuberculosis if the patient has had fever twenty-four hours before the blood has been taken, and a negative test is therefore said to be of more value than a positive one.

In the last analysis, no lesion can be diagnosed with absolute certainty as tuberculous until the eye is removed and sectioned and the histologic picture of tubercle noted, or until inoculation of tissue into animals succeeds in producing tuberculosis. Some pathologists may even question the histologic picture of tubercle unless the bacilli can be demonstrated in the lesion. It must be admitted, therefore, that a clinical diagnosis of tuberculous uveitis must be made *ex cathedra*.

From the Hospital of the University of Pennsylvania.

1. Bredeck, quoted by Post, M. H.: J. Tennessee M. A. **29**:179, 1936.
2. Williamson-Noble, F. A.: Brit. M. J. **2**:907 (Nov. 7) 1936.

Statistics vary as to the incidence of tuberculosis of the uveal tract in different clinics. In Germany one clinic [3] has reported that in as many as 50 per cent of the cases of iritis observed by them the condition was tuberculous, while in this country Irons and Brown [4] estimated that in less than 5 per cent of the cases of iritis observed by them the condition was caused by the tubercle bacillus. Tuberculosis, in its various forms, is not correspondingly more prevalent in Germany than in the United States. The discrepancy in these figures merely indicates that German ophthalmologists make this *ex cathedra* diagnosis more frequently than ophthalmologists in this country. This is true not only of tuberculous iritis but also of tuberculosis of the rest of the uveal tract.

There are at least two reasons why this diversity of opinion exists. Much more attention has been given in this country to other focal infections than to tuberculosis as the probable cause of lesions of the uveal tract, and there is no typical picture of tuberculous uveitis by which one can recognize this condition. The typical picture of tuberculosis, wherever it occurs in the body, is that of discrete nodular lesions, i. e., tubercles. These may be miliary or may coalesce to form large conglomerate tubercles, but in any case the essential feature is that of a well circumscribed nodular lesion.

When nodules are found on the surface of the iris during the course of iritis, one naturally thinks of tuberculosis, together with syphilis and other diseases in which nodules occur. If the iritis appears in the form of a plastic exudate, with no nodular lesions present, many clinicians in this country are not willing to consider a diagnosis of tuberculosis. Such cases are usually studied carefully from the standpoint of focal infections, and little attention is given to other causes once a negative serologic test has ruled out syphilis.

Prior to the time of von Michel it was generally believed that the only tuberculous diseases of the eye were those associated with the formation of tubercles. Von Michel [5] was the first to recognize the fact that, in spite of the absence of any macroscopic tubercles, the histologic sections of the iris in some cases of iritis showed miliary lesions which could be identified as true tubercles.

In this country one of the first to call attention to the possibility of tuberculosis in such cases was Verhoeff,[6] who reported a case of

3. Gilbert, W., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 3.

4. Irons, E. E., and Brown, E. V. L.: Recurrence of Iritis as Influenced by the Removal of Infections, J. A. M. A. **87**:1167 (Oct. 9) 1926.

5. von Michel, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 38.

6. Verhoeff, F. H.: Acute Tuberculous Iritis: Microscopic Examination of Eye Showing This Condition, Tr. Sect. Ophth., A. M. A., 1930, p. 21.

iritis the chief characteristic of which was acute fibrinous exudation with no formation of nodules. Tuberculosis was not suspected until the eye was removed because of excessive pain. Microscopic examination revealed the presence of characteristic tubercles in the rest of the uveal tract.

In the same manner, discrete miliary nodules seen in the choroid immediately suggest the possibility of tuberculosis, whereas in the forms of choroiditis in which the lesions are diffuse and highly exudative the condition is not likely to remind one of the possibility of this disease.

In 1932 Eggston [7] stressed the fact that tuberculosis may not always occur in the body in the characteristic form of tubercles. He pointed out that many conditions, undoubtedly of a tuberculous nature, are to be considered the expression of an allergic response on the part of the body to tuberculoprotein circulating in the blood, i. e., serous pleurisy, the cutaneous tuberculids and various ocular lesions, in which neither tubercles nor the tubercle bacilli could be demonstrated.

This conception of allergic tuberculous lesions is based on considerable experimental evidence. An excellent résumé of the work on this subject has been given by Allan Woods.[8]

EXPERIMENTAL PATHOLOGY

When a tuberculous infection is established in the body certain reactions take place in both the cellular and the humoral mechanisms. Perhaps the first defensive mechanism which is called into play is the cellular, and its expression is the collection of a group of lymphocytic cells around the invading organisms. If the bacilli are viable, these cells persist and change their character to that of epithelioid cells. Long and Holley [9] have shown that the large mononuclear cells, which ultimately give rise to epithelioid cells, come either from the blood stream directly or from the walls of the new ingrowing blood vessels, and not from the local fixed tissue cells, at least in the case of the cornea. If the bacilli are dead, no epithelioid cells may be formed, and the invading mononuclear cells may disappear, probably going back to the blood stream from which they came. Assuming that the invasion of bacteria is sufficiently virulent, a fight now takes place between the epithelioid cells and the bacteria. Giant cells form from the epithelioid cells. Some of the bacteria are engulfed and destroyed, while some of the cells

7. Eggston, A. A.: The Use of Tuberculin in Diagnosis and Treatment in Ophthalmology, Arch. Ophth. **8**:671 (Nov.) 1932.

8. Woods, Allan: Allergy and Immunity in Ophthalmology, Baltimore, The Johns Hopkins Press, 1933, p. 98.

9. Long, E. R., and Holley, S. W.: Am. J. Path. **9**:337, 1933.

are killed and form cellular detritus in the form of caseating material. If the bacteria succeed in breaking down this primary wall of defense they get into the blood stream and are carried either by the blood vessels or by the local lymphatics to fresh sites, and the infection spreads.

At the same time that the cellular defensive mechanism is doing its part, certain less well known humoral defenses are being formed. The nature of the substances in the blood stream is not well known. Antitoxin apparently does not exist. On the other hand, complement-fixing antibodies and agglutinins have been reported by a number of authors.[10] Once a tuberculous infection is established in the body, it is found that that body is more resistant to a second experimental infection. This increase in resistance has been called immunity.

When a subject whose immunity to tuberculosis has been increased is given a further injection of tubercle bacilli, either alive or dead, he may show a prompt and acute reaction, which is due to the sensitization of his tissues to the products of disintegration of the bacilli which caused the primary infection. This is called an allergic reaction. The subject has become sensitive, or allergic, to tuberculoprotein, and when he is further exposed to tuberculoprotein the result is a violent inflammatory exudative reaction.

Allergy does not occur with every infection of tuberculosis. If the infection is massive, the body may not respond by becoming allergic but may succumb before this stage can be reached. Further, allergy diminishes in the moribund patient suffering from tuberculosis. One group of investigators have expressed the belief that the allergic reaction is responsible for the development of immunity and that the two processes are really one and the same.[11] A second group have maintained that immunity is due not to the allergic reaction but to some other unknown factor, while allergy is an independent sensitization of the tissue cells to tuberculoprotein.[12] This school believes that the formation of miliary tubercles in the body, as a manifestation of tuberculosis, results when the number and virulence of the invading bacilli are high and the immunity low. If the body is allergic at the same time the result of such an infection may not be generalized miliary tuberculosis but inflammatory lesions of an extensive exudative, even necrotizing, type. This will be referred to in considering the various forms of uveal tuberculosis.

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10. Kunz, E.: Arch. f. Augenh. **109**:709, 1936.

11. Vaughn, W. T.: J. Lab. & Clin. Med. **21**:629, 1936.

12. (a) Paretzky, M.: Am. Rev. Tuberc. **31**:499, 1935; **33**:370, 1936. (*b*) Friedenwald, J. S., and Dessoff, J.: Bull. Johns Hopkins Hosp. **57**:148, 1935.

The difference between allergy and immunity has been clearly expressed by Rich and McCordock.[13] They defined allergy as a change in the body as a result of which the cells are more extensively damaged or killed by a given amount of tuberculoprotein than are similar cells in a normal body. Immunity, on the other hand, consists in the ability of an infected animal to hold locally tubercle bacilli introduced into the body, i. e., to prevent the bacilli from spreading from the site at which they first lodge, and, further, to prevent them from multiplying as rapidly as they would in the normal body. According to these authors, the bacilli must be held or fixed locally in the immune tuberculous animal by means of some specific precipitin-like substance which does not interfere with the free movement of other particulate matter.

The experiments of Friedenwald [14] support the belief of Rich and McCordock, that allergy and immunity are two different processes, independent of each other. Friedenwald was able to desensitize tuberculous guinea-pigs by giving repeated doses of tuberculoprotein over a long period. These desensitized animals showed no diminution in their resistance to reinfection, as judged by the mortality of the disease, the spread of the infection and the progress of the local lesion. In all these respects the desensitized animals showed greater resistance to reinfection than did the nondesensitized animals. This indicates strongly that allergy in tuberculosis cannot be regarded as essential to immunity. These results have been confirmed by Selter and Weiland.[15]

CLINICAL ASPECTS OF TUBERCULOUS UVEITIS

It is evident from what has been said that lesions caused by the tubercle bacillus may vary in their appearance and course, depending on such factors as the number and virulence of the infecting organisms, the state of immunity of the infected body and the degree of allergy of that body to tuberculoprotein. It is of interest now to review the various forms of uveitis and to see whether they can be explained on the basis of these variable factors.

Tuberculosis of the uveal tract is the most frequent form of ocular tuberculosis. It is practically always secondary to a tuberculous lesion elsewhere in the body and develops only in the second stage of tuberculosis, i. e., when a tuberculous nodule breaks down and the bacilli get into the blood stream. Ocular tuberculosis represents a metastasis, therefore, of tuberculous bacillemia. Once they get into the blood stream the bacilli are carried to the uveal tract and deposited there. Rarely

13. Rich, A. R., and McCordock, H. A.: Bull. Johns Hopkins Hosp. **44**:273, 1929.

14. Friedenwald, J. S.: Tr. Am. Ophth. Soc. **30**:269, 1932.

15. Selter, H., and Weiland, P.: Ztschr. f. Tuberk. **74**:161, 1935.

they may reach the uveal tract by way of the lymphatics or by direct extension of a tuberculous lesion from contiguous structures. Even in tuberculous choroiditis secondary to tuberculous meningitis the infection occurs by way of the blood stream, as Tooke [16] has recently shown, and not by spreading down the meninges.

If the ocular metastasis occurs shortly after the primary infection, before the body has become highly allergic, one should find the development of discrete miliary tubercles with little inflammatory reaction. Actually, the cases of tuberculous iritis which one sees clinically, in which there are many nodules in the iris and little inflammatory reaction, occur in young persons. There is little flushing of the ciliary body or pain, and not many precipitates are present on the posterior surface of the cornea, but studded here and there in the stroma of the iris are discrete yellowish or gray nodules which often disappear in the course of a few days or weeks, leaving small areas of atrophy. Often these nodules are too small to be seen except with the aid of magnification such as is possible with the slit lamp; this should be used in every case of iritis and a careful search made for these miliary lesions. Although the larger nodules seem to have a predilection for the ciliary margin of the iris, they occur frequently near the pupillary edge, and it is here that the smaller ones are most frequently found. This form of tuberculous iritis occurs in young persons from 10 to 25 years of age. The subjects frequently have a history of tuberculous contacts. They sometimes show definite pulmonary lesions on roentgen examination but are not likely to have any marked sensitivity to tuberculoprotein.

In the posterior portion of the uveal tract this type of iritis is best represented by the miliary tubercules of the choroid occurring in children with tuberculous meningitis. These are the easiest types to diagnose as tuberculous because of the frankly nodular character of the lesions.

In the next decade one finds a type of iritis more highly inflammatory in character, but with little tendency to the formation of nodules. If nodules are present, they are likely to be fewer. There are considerable pain and some plastic exudate, and the iris quickly becomes bound down with posterior synechia. This condition is frequently associated with a corneal condition, such as sclerosing keratitis. In the posterior segment this inflammation is represented by large greenish gray lesions, usually single, in the extreme periphery of the choroid. The vitreous is so hazy that often the details of the fundus cannot be made out, but careful search in the periphery reveals the characteristic white or greenish color of the underlying lesion against the dark or red background. The patients, ranging in age from about 20 to 30 years, usually have

16. Tooke, F. T.: Tr. Am. Ophth. Soc. **33**:201, 1935.

a high degree of cutaneous sensitivity to tuberculoprotein and rarely show any active lesions in the lungs. The roentgen rays commonly show old healed lesions at an apex and enlarged peribronchial lymph glands.

Patients from 30 to 50 years of age show the next most common type of tuberculous uveitis met with. In these the condition is seldom iritis alone; the ciliary body is equally involved. The lesions are distinctly highly exudative. The whole posterior surface of the cornea is covered with mutton fat deposits. There is little flushing of the ciliary body or pain, but the whole iris is firmly plastered down to the lens when the patients first come for examination because of failing vision. If the pupil can be dilated the vitreous is found to be full of opacities which prevent any view of the fundus. In these cases the condition is usually bilateral and is a frequent cause of blindness. If secondary glaucoma does not occur, because of the occlusion of the pupil, the globe generally becomes phthisical. A large percentage of these cases are found in the colored race. Nearly all the patients have a high degree of allergy. Clinical and roentgen examination of the chest reveals about the same picture as in the previous type of uveitis.

TABLE 1.—*Characteristic Features of Tuberculous Iritis*

Age	Type of Lesion	General Tuberculosis	Allergy*
10 to 20	Nodular discrete tubercules; little inflammation or exudate	Occasionally active lesions	Very little
20 to 30	Acute plastic iritis; few, if any, nodules; highly inflammatory; moderate exudation	Seldom active but healed lesions	Moderate
30 to 50	Chronic iritis; marked exudate; little inflammation; mutton fat deposits; occasional large nodules	Healed lesions	Marked

* As will be explained in the section on the Mantoux test, allergy is a quantitative matter. In our own tests we considered a patient as moderately allergic if he reacted positively to 0.0001 mg. of purified protein derivative. If he did not have a positive reaction to 0.01 mg., he was not considered allergic.

There is no characteristic lesion of the posterior segment of the uveal tract which corresponds to this type of iridocyclitis. Many of the German writers include in this category the various forms of disseminated choroiditis seen in patients at this age. At present the evidence seems insufficient to classify them as such; the lesions are certainly not highly exudative, and the condition seems to us to be more a degenerative than an inflammatory process. Perhaps tuberculosis should be considered, but it would be reprehensible to try to provoke a focal reaction with tuberculin for diagnostic purposes.

The tables summarize the characteristic features of the various forms of iritis and choroiditis commonly seen.

RELATIONSHIP OF OCULAR TUBERCULOSIS TO GENERAL TUBERCULOSIS

Recent statistics show that almost 100 per cent of the adult population of the large cities of this country have had at some time during their life an infection with the tubercle bacillus, and this infection has been sufficient to give them a cutaneous sensitivity to tuberculoprotein.[17]

This would seem to indicate that ocular tuberculosis should be fairly common, especially among those with active pulmonary lesions, and yet the reverse is true. It is generally recognized that patients with ocular tuberculosis seldom have active pulmonary lesions and that ocular tuberculosis is extremely rare in sanatoriums for patients with pulmonary tuberculosis. Of a thousand patients with active pulmonary tuberculosis examined by Goldenburg and Fabricant,[18] in only three could the ocular condition be considered as due to tuberculosis, and only

TABLE 2.—*Characteristic Features of Tuberculous Choroiditis*

Age	Type of Lesion	General Tuberculosis	Allergy
1 to 10	Miliary tubercles of choroid	Tuberculous meningitis	Very little
10 to 30	Inflammation of the anterior part of the uvea; single large exudative lesion; little inflammation; tuberculous periphlebitis	Healed lesions	Moderate or severe
30 to 50	Disseminated choroiditis included by many German writers	Healed lesions	Not stated in the literature; our own results did not indicate any constant degree of allergy

nineteen patients showed any pathologic changes in the posterior segment of the globe which could have been tuberculous. This paradox has never been satisfactorily explained, but we believe it is no proof of the claim made by these authors that tuberculosis of the eye must be much less common than the reports in the literature indicate.

In clinics where many patients with suspected ocular tuberculosis are seen the consensus is that the vast majority show healed pulmonary lesions or lesions of the peribronchial lymph glands.[19]

Axenfeld and de la Camp were never able to demonstrate any active lesions in the lungs of their patients by the roentgen rays, and these

17. Hetherington, H. W.; McPhedran, F. M.; Landis, H. R. M., and Opie, E. L.: Tuberculosis in Medical and College Students, Arch. Int. Med. **48**:734 (Nov.) 1931; Further Study of Tuberculosis Among Medical and Other University Students, ibid. **55**:709 (May) 1935.

18. Goldenburg, M., and Fabricant, N. D.: Eye in Tuberculous Patient, Tr. Sect. Ophth., A. M. A., 1930, p. 135.

19. Werdenberg: Klin. Monatsbl. f. Augenh. **75**:545, 1925. Axenfeld, T.: ibid. **85**:465, 1930.

negative findings were confirmed by Wendt.[20] Werdenberg, on the other hand, found no person among one hundred and thirty patients examined in whom the lungs did not show some pathologic changes. Ninety of these patients showed moderate involvement, and thirty showed advanced tuberculosis of the apexes or at the hilus. Gilbert [21] also claimed that in practically every case of tuberculous uveitis the roentgen rays reveal some evidence of pulmonary tuberculosis.

THE MANTOUX TEST

As has been indicated, accurate diagnosis of uveal tuberculosis is difficult. Among the most important of the diagnostic aids is the intracutaneous tuberculin test, or the Mantoux test. When properly performed it is of great help in establishing the presence of cutaneous sensitivity to tuberculoprotein.

In the proper performance of the test certain precautions should be observed and certain errors avoided. The recommendations are as follows:

Method.—The intradermal test is rapidly supplanting other tuberculin tests, because the dosage can be accurately controlled and in that way undesirable local, focal and constitutional reactions can be avoided. These are particularly apt to occur in subcutaneous and Calmette (ophthalmic) tests.

Materials.—Many kinds of tuberculin have been tried. A potent old tuberculin is very satisfactory, but because of the variability in strength of different batches efforts have been made to produce a stable uniform product. To this end a purified protein derivative of the tubercle bacillus has been produced and tested by Seibert, Aronson, Reichel, Clark and Long. This promises to supplant old tuberculin because it is potent and its strength varies little in different batches. Test dilutions should be made up fresh at least once a month and refrigerated.

Syringes.—Each syringe should be reserved for its own strength of old tuberculin or purified protein derivative, to avoid false positive reactions.

Dosage.—As a high percentage of the population, especially the urban population, have sensitivity of the skin to tuberculin in high concentration,[17] it would seem important to determine the degree of sensitivity rather than its mere presence.

To this end the following schedule is suggested: The initial dose should be subminimal (about 0.000001 mg. of purified protein deriva-

20. Wendt, H.: Klin. Monatsbl. f. Augenh. **71**:686, 1923.

21. Gilbert, W., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 42.

tive). With each negative reaction the dose is increased tenfold until a positive reaction is obtained. The resultant positive reaction is reported as +1, +2, +3, or +4, depending on the size and severity of the reaction (as suggested by Aronson), and the dose is stated. In this manner an accurate gage of the patient's degree of sensitivity is obtained. This test is called the quantitative Mantoux test (Ayman [22]) and should supplant what might be called the qualitative test. A true positive Mantoux test indicates a sensitivity to tuberculoprotein. This can only follow a tuberculous infection. It does not indicate activity or quiescence of a general lesion or the presence or lack of immunity. However, researches by Friedenwald and Dessoff [12b] indicated that patients with ocular tuberculosis exhibit a higher degree of cutaneous sensitivity to tuberculoprotein than patients with nontuberculous ocular lesions.

TREATMENT OF OCULAR TUBERCULOSIS

The treatment of ocular tuberculosis may be divided into prophylactic and curative measures.

Prophylactic measures, aimed to prevent the occurrence of ocular tuberculosis, necessarily fall in line with the widespread campaign to eradicate general tuberculosis. These measures may be specific or general.

The specific prevention of tuberculosis is being attempted on a wide scale through the use of Bacillus Calmette-Guérin (BCG). This is an attenuated tubercle bacillus which is given by mouth or hypodermically to induce specific immunity. There is much experimental evidence to indicate its efficacy, but its general adoption has been rather slow, probably because of tragic events associated with its early use. It has been used more extensively in Europe than in America.[23]

Smithburn, Sabin and Geiger [24] called attention to the unpleasant possibility, in using tuberculin M. A. 100,[25] of increasing the patient's

22. Ayman, D.: The Intracutaneous Quantitative Tuberculin Test, J. A. M. A. **103**:154 (July 21) 1934.

23. Clawson, B. J.: Experiments Relative to Vaccination Against Tuberculosis with the Calmette-Guérin Bacillus (BCG), Arch. Path. **20**:343 (Sept.) 1935. Scheel, O.: Rev. de la tuberc. **1**:529, 1935. Nègre, L., and Valtis, V.: Bull. Acad. de méd., Paris **113**:585, 1935.

24. Smithburn, K. C.; Sabin, F. R., and Geiger, J. T.: Am. Rev. Tuberc. **29**:562, 1934.

25. Tuberculin M. A. 100 is a product which was developed by Funk and Huntoon. Tubercle bacilli are grown on a special synthetic nonprotein medium. The only protein in the filtrate of such a culture is therefore synthesized by the tubercle bacilli and represents pure tuberculoprotein. The protein precipitated by ammonium sulfate is called M. A. 100. (Funk, E. H., and Huntoon, F. M.: Biochemical Studies of Bacterial Derivatives: Skin Reactions in Man with Human Tubercle Bacillus Protein M. A. 100; Preliminary Report, J. Immunol. **19**:237 [Aug.] 1930.)

sensitivity to tuberculoprotein without affording any immunization to tuberculosis.

Curative treatment of ocular tuberculosis, too, may be divided into general and specific measures.

The general measures are very important, though often neglected.

The importance of rest was emphasized by E. V. L. Brown,[26] who reported four cases of tuberculosis of the anterior segment complicating active pulmonary tuberculosis. The disease healed seemingly by treatment with rest alone.

Adequate diet, rest, heliotherapy and fresh air are as much indicated in the care of a patient with ocular tuberculosis as in the case of a patient with general tuberculosis, and in many cases the condition fails to improve until these measures are applied.

Many physical therapeutic measures are employed, among which are diathermy and the use of the infra-red rays, hot pads and the roentgen rays. Favorable reports have been made following their use when properly applied in selected cases. Therapy with nonspecific foreign proteins, such as milk, typhoid vaccine and other vaccines, and diphtheria antitoxin, has a place in certain cases, and its use has been recently discussed by F. H. Newton.[27]

Autohemotherapy, too, has its advocates. Good results have been reported of injections of blood into the anterior chamber in ocular tuberculosis, especially from abroad.[28]

In addition to the use of the well established and accepted drugs certain newer pharmacologic measures have been recommended in many reports.

Gold is being used as gold sodium thiosulfate or a double thiosulfate of sodium and gold, given intravenously, especially in cases in which the patient has not responded to tuberculin therapy.[29]

Neoarsphenamine [30] is being used with success, even in nonsyphilitic uveitis. It is recommended for treatment of patients whose condition has an obscure etiology and has not responded to other types of treat-

26. Brown, E. V. L.: Am. J. Ophth. **19**:668, 1936.

27. Newton, F. H.: Empirical Treatment of Uveitis, Arch. Ophth. **14**:618 (Oct.) 1935.

28. Miranda, A. Garcia: Arch. de oftal. hispano-am. **34**:349, 1934. Serr, H.: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **50**:41, 1934. Denig, Rudolf: Injection of Patient's Own Blood into Anterior Chamber for Tuberculous Iritis, Arch. Ophth. **14**:860 (Nov.) 1935. Kyrieleis, W.: Ztschr. f. Augenh. **85**:16, 1934.

29. Leoz, G.: Arch. de oftal. hispano-am. **35**:230, 1935.

30. Lucic, Hugo: Neoarsphenamine in the Treatment of Nonsyphilitic Inflammations of the Uveal Tract, Arch. Ophth. **15**:826 (May) 1936.

ment. Mercury and iodides, too, have their place, but the iodides must be used cautiously, if at all, for fear of reactivating old pulmonary lesions.

Specific treatment consists in the use of tuberculin. Many different forms have been suggested, but old tuberculin is probably the most widely used. Other preparations of tuberculin are as follows:

Denys bouillon filtrate,[31] preferred by Gay [32] and his colleagues at the Wilmer Ophthalmological Institute.

Bacillen emulsion (B. E.).

Tuberculin A. O., the Japanese tuberculoprotein.

Tuberculin M. A. 100, developed by Funk and Huntoon.

Beraneck's tuberculin.[33]

Although tuberculin is generally given hypodermically, it has been suggested that it be given by the percutaneous route by scarification with glass or emery paper.[33a]

The interval between hypodermic injections should be from four to seven days. The initial dose may safely be one tenth of the amount of tuberculin necessary to elicit a positive cutaneous reaction. Subsequent doses may be increased by 10 to 100 per cent to a maximum of from 50 to 100 mg. of old tuberculin or its equivalent. Severity and persistence of local reactions serve as a guide to the frequency and the size of the dose.

It is generally conceded that a definite focal ocular reaction is a serious menace to the integrity of an affected eye and calls for extreme caution in administering subsequent injections, which should be radically reduced in amount and increased later only with circumspection.

Some ophthalmologists, however, boldly seek a dose sufficient to elicit a slight focal reaction in the belief that this inflammatory reaction is necessary to fix the bacillus and heal the lesion.[34]

However, this view is not shared by most ophthalmologists, who seek to avoid gross focal reactions. They attribute the beneficent effect of tuberculin therapy either to subclinical microscopic focal inflamma-

31. Denys: Le boüillon filtré, Paris, Uystpruys-Louvain, 1925.

32. Gay, Leslie N.: The Treatment of Ocular Tuberculosis with Tuberculin, Arch. Ophth. **3**:259 (March) 1930.

33. Beraneck's tuberculin is derived from a glycerinated bouillon made without peptone. This medium is less rich in albuminous substances than mediums containing peptone and hence less likely to cause false positive reactions. The tuberculin is a combination of the exotoxins from the filtrates and endotoxins extracted from the ground bacilli by a 1 per cent solution of orthophosphoric acid. (Sutherland, H. G.: Control and Eradication of Tuberculosis, Edinburgh, William Green & Son, 1911, pp. 105-118.)

33a. Löwenstein, A.: Ztschr. f. Augenh. **85**:191, 1935.

34. Samojloff, A., and Tihomirova, A.: Ann. d'ocul. **172**:993, 1935.

tory reactions or to the desensitization of the cells of the affected area so that they no longer are irritated by contact with tuberculoprotein.[12b]

For the malignant exudative forms of ocular tuberculosis it is deemed unwise by some to employ any tuberculin therapy at all, because of the danger of focal damage.[35]

Under these conditions only conservative local and general treatment should be employed.

There are, too, investigators who raise the question whether the good effect following the use of tuberculin may be nonspecific rather than due to specific desensitization or immunization.[36]

SUMMARY

Tuberculous lesions of the uveal tract vary greatly in their appearance and clinical course. The lesions of the iris may be nodular or diffuse. There may be marked inflammatory reaction with severe pain, flushing of the ciliary body and synechia, or the eye may be relatively quiet. There may be little exudate, or the whole posterior surface of the cornea and the floor of the anterior chamber may be filled with mutton fat deposits.

The choroidal lesions may likewise exhibit great variance in the aforementioned characteristics.

It is possible to group patients with tuberculous uveitis into three types: those whose lesions are chiefly nodular, those whose lesions are chiefly inflammatory and those whose lesions are mainly exudative. Lesions of the nodular type occur chiefly in the young up to 20 years of age; those of the inflammatory type, in persons from 20 to 30 years of age, and those of the exudative type, in persons from 30 to 50 years of age. The allergy increases in degree in a general way in the order mentioned and is the factor responsible for changing the typical picture of tubercle to that of a less characteristic inflammatory or exudative uveitis. Every patient with uveitis should be studied from the standpoint of tuberculosis. Healed lesions of the chest should be searched by the roentgen rays and a quantitative Mantoux test should be done with purified protein derivative to determine the degree of allergy.

35. Werdenberg: Useful and Harmful Therapeutics in Tuberculosis, Arch. Ophth. **13**:303 (Feb.) 1935.

36. Lockwood, Chester: Am. J. Ophth. **19**:156, 1936.

News and Notes

Advisory Board for Medical Specialties.—The annual meeting of the Advisory Board for Medical Specialties, which is the coordinating board of the twelve certifying boards in the various specialties, the Association of American Medical Colleges, the American Hospital Association, the Federation of State Medical Boards of the United States and the National Board of Medical Examiners, was held at Atlantic City, N. J., on June 6, 1937. The following officers and executive committee were elected:

Willard C. Rappleye, M.D., president, New York
W. P. Wherry, M.D., vice president, Omaha
Paul Titus, M.D., secretary-treasurer, Pittsburgh
W. B. Lancaster, M.D., Boston
R. C. Buerki, M.D., Madison, Wis.

Dr. Louis B. Wilson, of Rochester, Minn., the retiring president, was elected an emeritus member of the board.

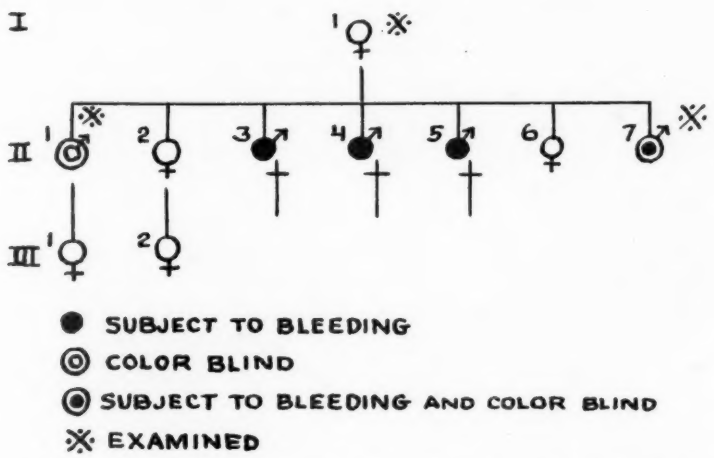
Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Color Sense

HEMOPHILIA AND COLOR BLINDNESS OCCURRING IN THE SAME FAMILY. W. J. B. RIDDELL, Brit. J. Ophth. **21:** 113 (March) 1937.

The pedigrees of seven hemophilic families were investigated as far as practicable, and the affected members were examined for color defects. Ishihara's isochromatic plates were used in all cases, but additional tests were used for confirmation. Only one pedigree of a family



The pedigree of a family in which one member showed color blindness associated with hemophilia.

which showed color blindness associated with undoubted hemophilia was obtained and this was obtained from a dental clinic. The relative position of this pedigree is shown in the diagram.

W. ZENTMAYER.

Comparative Ophthalmology

THE CHEMISTRY OF THE RETINA. A. C. KRAUSE, Am. J. Ophth. **19:** 555 (July) 1936.

Krause describes his experimental methods and presents a table showing the chemical constitution of the bovine retina. He comments on the various constitutents and gives the following summary:

"The chemical constitution of the bovine retina has been determined. Most of the proteins are complex lipid-proteins of nervous-tissue origin. The amount of supporting-tissue proteins is relatively small."

W. S. REESE.

MICROANATOMY OF THE EYE WITH THE SLITLAMP MICROSCOPE. M. U. TRONCOSO and R. CASTROVIEJO, Am. J. Ophth. **19:** 583 (July) 1936.

This is the last of a series of articles. It describes the details of the angle of the anterior chamber in primates and in man. The authors then comment on their work and that of other investigators and give a table of measurements found in ungulata, carnivora, monkeys and man.

W. S. REESE.

Congenital Anomalies

BILATERAL PUPILLARY ANOMALIES; PERSISTENCE OF THE ANNULAR SINUS. V. VILLARD and H. VIALLEFONT, Bull. Soc. d'opht. de Paris, April 1936, p. 317.

A girl of 13 years had noted a peculiar appearance of her eyes for many years. On examination one is impressed by an anomaly of the pupil and the iris. Externally the chestnut-colored iris was perfectly normal, but the internal portion, which was seen best with the slit lamp, showed a veritable border about 0.3 to 0.5 mm. wide, forming a complete circle of blackish brown, the classic color of the uveal pigment. At a distance this anomaly made the eyes appear as though the pupillary borders were markedly irregular, the usual curved pupil appearing serrated. Photographs and drawings of the condition are shown. Szili and Gallenga have reported like conditions. The anomaly is evidently caused by a developmental failure of the pigment of the iris to cover that of the uvea.

L. L. MAYER.

Cornea and Sclera

MEGALOCORNEA. M. U. TRONCOSO and I. E. GIVNER, Am. J. Ophth. **19:** 549 (July) 1936.

Troncoso and Givner define and discuss the etiologic theories of megalocornea and report a case of this condition in a 28 year old man who was studied thoroughly. The following summary is given:

"1. As a basis for megalocornea, disturbed endocrinology is not supported by the accepted laboratory procedures of the day.

"2. The considerable stretching found in the ciliary body and iris is not in accord with the theory of overdevelopment of the entire eyeball nor even of the anterior segment in its entirety, for the size and relations of the structures of the angle were not proportional to the enlargement of the cornea.

"3. The gonioscopic findings do not give support to the theory of drainage obstruction, at least not by peripheral synechia. On the other hand, the presence of hypotony in this and other cases in the literature point to either diminished outflow or atrophy of the ciliary body due to stretching.

"4. Cases of megalocornea presented in the future should be tabulated giving specific data concerning endocrinology, X-ray, and gonioscopic findings."

W. S. REESE.

SCLEROKERATITIS, PROBABLY BACILLARY. F. TERRIEN and P. HALBRON, Bull. Soc. d'opht. de Paris, December 1936, p. 772.

The reason for the presentation of this patient was the good result obtained by general treatment.

A woman 25 years old had sclerokeratitis of the left eye of three weeks' duration. There existed pericorneal injection and mild infiltration of the cornea, with a small area of elevation. With local treatment it was noted that the condition extended. Four small points of scleral infiltration appeared between 3 and 4 o'clock near the limbus. General examination showed no tuberculosis, although a picture of the lungs showed perihilar shadows. The basal metabolic rate was + 13.5 per cent. Successive injections of 0.1, 0.2, 0.25, 0.5, 0.75 and 1.0 cc. of methylic antigen were given. After some time visual acuity returned to normal, all vascular activity near the cornea ceased, and the slit lamp showed only slight infiltration of the corneal limbus, with absence of blood vessels. Terrien and Halborn believe that because of the course and recovery the diagnosis of tuberculous sclerokeratitis may be presumed.

L. L. MAYER.

ETIOLOGY AND TREATMENT OF KERATOCONJUNCTIVITIS SICCA. E. VON GROSZ, Klin. Monatsbl. f. Augenh. **97**: 472 (Oct.) 1936.

Sjögren's syndrome is characterized by the following symptoms: dry keratoconjunctivitis, xerostomia and dry pharyngolaryngitis. These symptoms are caused by atrophy, or deficiency of secretion, respectively, of the glands. The disease occurs chiefly in women undergoing the menopause or suffering from arthritis. The findings in the cases observed by von Grosz showed that the source is not an infection but is a disease of the blood-forming organs. Characteristic signs of pernicious anemia were found in two typical cases, which suggests a connection between this type of anemia and the syndrome. Careful examination of the blood is essential in these cases. The menopause and arthritis are both caused by dysfunction of the ovaries; the condition is brought on by a disturbance of the vitamin metabolism, in the author's opinion. Study of a larger series of cases may substantiate this view. Aside from the ophthalmologic interest, the syndrome furnishes equally interesting problems for the internist, on account of the pernicious anemia; for the stomatologist, because of the xerostomia, and for the rhinologist, on account of the ozena. Therapeutic possibilities are the use of estrogenic substance, injections of acetylcholine and antianemic treatment with liver, iron and arsenic. Other therapeutic agents are vitamins A, B and C. For local application are mentioned white of the egg, fibrolysin and oil containing vitamin A.

K. L. STOLL.

Experimental Pathology

RESPONSE OF THE PUPILLODILATOR AND PUPILLOCONSTRICTOR NERVES TO MYDRIATICS AND MIOTICS IN EXPERIMENTAL HYPOCALCEMIA AND HYPERCALCEMIA. F. CASINI, Arch. di ottal. **43**: 231 (Dec.) 1936.

The literature concerning the effects of calcium on the vegetative nervous system is reviewed. Hypocalcemia was induced in rabbits by

intravenous injections of a 1 per cent solution of sodium oxalate and a 20 per cent solution of sodium [illegible]ride. Solutions of pilocarpine, epinephrine hydrochloride and cocaine which were effective in normal animals were ineffective in animals with hypocalcemia produced in this way. Miosis and mydriasis could still be produced, but only with more concentrated solutions. Hypercalcemia was produced in other rabbits by intravenous injections of calcium gluconate, the excess of calcium of the blood being from 1 to 2 mg. per hundred cubic centimeters. In these animals the pupillary response to drugs remained normal. Casini concludes that lack of calcium has a depressing effect on the endings of both the dilator and the constrictor nerves. The effect is the same, as was shown with respect to other nerves, whether the nerves are sympathetic or parasympathetic. The author believes this effect takes place by means of changes in the electrochemical equilibrium of the aqueous.

S. R. GIFFORD.

EXPERIMENTS ON NONPERFORATING CAUTERIZATION OF THE SCLERA. A. DE SANCTIS, Arch. di ottal. **43**: 252 (Dec.) 1936.

The sclera in rabbits was treated with the actual cautery 13 mm. back of the limbus for one-half minute. The tip of the cautery was 1.5 mm. in diameter and was kept violaceous to avoid too rapid an effect. The eyes were examined ophthalmoscopically and histologically, being removed at periods varying from just after the operation to ten days later. The observations were similar to those reported by other investigators after coagulation by diathermy.

S. R. GIFFORD.

General

THE THERAPY AND PATHOLOGY OF PURULENT OCULAR DISEASES. N. BLATT, Orvosi szemle **8**: 411, 1935.

In hardly any part of the organism may purulent diseases bring about such serious pathologic changes and loss of function as in the eye. Such changes, unless therapeutic measures are carried out thoroughly and in due time, may lead to total loss of the eyeball. The prognosis in many cases of ulcus serpens corneae, orbital phlegmon and purulent iridocyclitis is unfavorable. It is desirable to eliminate from the nomenclature the designation "idiopathic," since many times the primary and ignored cause of the iridocyclitis called idiopathic is inflammation of the gallbladder, influenza, a dental disease or suppuration of the tonsils.

Focal infections are generally admitted to be causative of purulent changes in the eyes. However, one should not be too eager to attribute the cause to focal infections but should practice rigorous criticism. It is necessary to consider the accessory nasal cavities and to have a rhinologic examination. One should, however, not exaggerate. If the primary cause is in one of the accessory nasal cavities one must not only obtain drainage of the cavity, but pay full attention to the eye and support the entire organism against the purulent infection.

Purulent foci, though located in the remotest parts of an organism, may produce metastases in the eyes. One must emphasize, in particular,

purulent diseases of the prostate. In the absence of tuberculosis, syphilis, diabetes, leukemia, acute fev injuries, dental disease, tonsillar infections and gastro-intestinal diseases, the technical term "idiopathic" is sometimes applied to keratitis, iritis and choroiditis when possibly the matter is a prostatic infection. One should never forget the causal rôle that disease of the prostate may play in the causation of purulent iridocyclitis.

Puerperal fever may also be causative of serious purulent iridocyclitis, and the latter may in turn be complicated, even fatally, by orbital phlegmon, thrombosis of a sinus and cerebral abscess.

Exogenous suppuration arising from an injury of the eye is likewise known as a possible cause of central fatal complications. Failure of all means of treatment has often been recorded. No success has been obtained in treatment with serums, but protective smallpox inoculation combined with subsequent injections of milk and a combination of turpentine, quinine, ethyl aminobenzoate and 84 per cent oil has proved beneficial.

Purulent iridocyclitis occurring from failure of an operation on an eye may offer the same picture as iridocyclitis due to an injury. Adequate treatment provides reopening of the wound of the operation, removal of the hypopyon, cauterization of the raw surfaces, subjunctival injection of a solution of mercury oxycyanide, treatment with serums against streptococci and pneumococci, use of ethylhydrocupreine hydrochloride as an eye lotion and intravenous injection of a colloidal suspension of silver, a preparation containing caffeine, sodium salicylate and methenamine, or the preparation of turpentine and quinine just mentioned.

As a matter of fact all the aforementioned purulent ocular diseases can heal (avoiding relapses) only if the primary focus (teeth, tonsils, prostate) is healed; the healing process is to be produced in concomitance with local treatment of the eye and general treatment. Injections of the turpentine and quinine preparation offer good results.

In seventy cases of suppuration of the eyes (purulent iridocyclitis, corneal abscess, purulent peridacryocystitis, orbital phlegmon and purulent infiltrations of the eyelids) the turpentine and quinine preparation was applied with satisfactory therapeutic results. Intragluteal injection of 1 cc. of this preparation every other day or every third day was not attended by harmful effects. Simultaneous repeated examinations of the urine are advisable, controlling possible irritation of the kidneys.

N. Blatt.

General Diseases

Ocular Manifestations in Trichinosis. E. Triandaf and J. Nitzulescu, Arch. d'opht. **53:** 47 (Jan.) 1937.

This report deals with the observation of three patients with trichinosis in one family. The symptoms were the same in all but varied in severity, the condition being acute and of long duration in one and mild and fleeting in the other two. The most characteristic symptom, which was present in all three cases, was edema of the lids and face, with an eosinophil count of from 17 to 18 per cent.

S. B. Marlow.

RUBEOSIS IRIDIS DIABETICA. O. KURZ, Arch. f. Augenh. **110: 284**, 1937.

In 1928 Salus reported on proliferation of the blood vessels in the iris of diabetic patients, which, because of its characteristics and constant association with secondary glaucoma, he classified as a definite symptom complex. Kurz reports two cases of such a condition. In one case one of the eyes had to be enucleated because of absolute glaucoma, and the author gives a description of the histopathologic changes in this eye. From his observations he has come to believe that rubeosis iridis consists in true proliferation of the blood vessels of both the pupillary margin and the ciliary region of the iris. The vessels arise in the stroma of the iris itself and not in any deposition of connective tissue. The proliferation of the blood vessels in the ciliary region leads to the formation of anterior synechiae, which are the cause of the secondary glaucoma. Diabetic retinitis is frequently accompanied with hemorrhages. These changes are most frequently seen in diabetic patients with arterial hypertension. The anatomic changes in the walls of the vessel are rather inconstant and are not specific. It is certain that thromboses, with the formation of a collateral circulation, are not the cause of this condition. Kurz believes that the formation of these vessels is an expression on the part of the tissues of an attempt to improve their damaged nutrition.

F. H. ADLER.

General Pathology

HISTOPATHOLOGIC STUDY OF CHANGES IN THE OPTIC NERVE IN PARESIS. A. BIFFIS, Ann. di ottal. e clin. ocul. **65: 1** (Jan.); 105 (Feb.) 1937.

The literature on the subject is carefully reviewed. Biffis examined autopsy material in twelve cases of paresis. The whole course of each optic nerve, the chiasm and adjacent portions of the frontal lobe were prepared for the usual histologic stains and for the various methods of gold and silver impregnation and special stains for the nervous elements. The methods which were found to be of most value are described. Fixation was aided by injection of a 10 per cent solution of formaldehyde U. S. P. into the orbit and vitreous chamber immediately after death. The findings of ophthalmoscopic examination were available in only a few cases. Signs of atrophy of the optic nerve were noted histologically in only two cases. In one of these peripheral degeneration of fibers was present and in the other a focus of central degeneration, affecting in each case the intracanalicular portion of the nerve. In the former cases an increase in oligodendroglial elements was present, especially in the tracts of fibers affected by atrophy. In the second case an increase of these elements was present along the whole course of the nerve, being less marked about the area of central degeneration. In the other ten cases the glia, microglia and oligodendroglia showed no distinctive changes.

In this respect paresis is seen to present a marked difference from tabes dorsalis, in which atrophy of the optic nerve is a common and early finding. In paresis the degenerative changes observed bore no

direct relationship to changes in the nerve sheaths. In the diffuse degeneration seen in one case, with involvement of microglia and oligodendroglia, the primary factor seems to have been sclerosis of capillaries supplying the septums.

S. R. GIFFORD.

Glaucoma

ACTION OF CARBAMINOYLCHOLINE ON THE NORMAL EYE AND THE GLAUCOMATOUS EYE. G. DE SANCTIS, Ann. di ottal. e clin. ocul. **65:** 25 (Jan.) 1937.

De Sanctis reviews the literature and reports his observations on the use of carbaminoylcholine. In normal eyes miosis was prompt, the diameter of the pupil reaching 1.5 mm. in an hour after instillation. This contraction was maintained for three or four hours, the pupil returning to normal in twelve hours. The ocular tension was reduced in normal eyes by 2 to 4 mm. of mercury, returning to normal within six to ten hours. One instillation was employed for the observations reported. Apparently the 0.75 per cent solution recommended by most authors was employed, although this is not stated. In two cases of absolute glaucoma no effect was noted on the tension, although in one case the pupil became slightly small. In one case of glaucoma secondary to hypermature cataract the tension was reduced only slightly by the drug but not to normal. In three cases of chronic simple glaucoma the effect on the pupil and on the tension was marked. The pupil contracted to 1.5 mm. in diameter within an hour, and the tension was reduced to normal within this time. The reduction of the tension following one instillation persisted for thirty to forty hours. The drug was effective in one case in which the effects of pilocarpine had been insufficient. Moderate hyperemia, with some pain in the eye and headache, was noted after the use of the drug.

S. R. GIFFORD.

Lens

EXTRACTION OF CATARACT IN 15,000 OPERATIONS. E. DE GRÓSZ, Arch. d'opht. **53:** 161 (March) 1936.

De Grósz describes the operative technic which has been developed in his clinic as the result of thirty years of observation and calls attention to the increasingly better results with the development of the new technic for producing anesthesia and the better selection of cases. He outlines the Török procedure for the intracapsular extraction of cataract. Iridectomy, he believes, is safer than attempting to obtain a round pupil. In thirty years the percentage of cases of extraction of cataract in which loss of vitreous occurred rose to 2.4. For the operations for extracapsular extraction it was 1 and for the operations for intracapsular extraction 4. Within more recent years it has been decreasing (it was 2.6 for 1923-1927 and 1 for 1928-1931). Infection has been decreasing (there was a decline from 1 per cent ten years ago to 0.4 per cent in 1932). Expulsive hemorrhage was observed twelve times in thirty years, or about once in 1,200 cases. Since the use

of retrobulbar injection it has occurred more frequently. The percentage for 14,207 cases in which there were no complications was 0.087, and for the cases in which there were complications it was 0.59. Since the use of retrobulbar injection the percentage has risen from 0.1 to 0.3. As a result de Grósz has been led to pay more attention to the vascular system than formerly.

S. B. MARLOW.

PREPARATORY IRIDECTOMY IN CAPSULOLENTICULAR EXTRACTION OF CATARACT. L. PAUFIQUE, Bull. Soc. d'opht. de Paris, February 1936, p. 115.

Paufique believes that total extraction of the lens in its capsule is the ideal operation for cataract. He has had good results in the past four years using the procedure of Arruga. This paper is a report of thirty cases of cataract in which, contrary to the usual technic, because of anticipated complications and difficulties preliminary iridectomy was done. In this group, in two cases there was glaucoma; in eight there was severe diabetes; in six there was very high myopia; in seven there were chronic iritis and pupillary seclusion, and in four, hypertension and albuminuria, and in three cases the cataract was in an otherwise normal eye, the other eye being blind. Preliminary iridectomy is done from three weeks to a month before extraction, after a small keratome incision is made near the limbus. The results in the thirty cases have been good. The advantages of this technic are as follows:

1. There is no prolapse of vitreous. 2. Extraction is easy—there is no rupture of the capsule, and the integrity of the hyaloid membrane is maintained. 3. There is no hemorrhage into the anterior chamber. 4. Most important, there is no prolapse of the iris.

L. L. MAYER.

CHANGES IN GLYCOLYSIS IN THE LENS DUE TO AGE. L. K. MÜLLER, Arch. f. Augenh. **110:** 206, 1936.

The experiments reported here were undertaken to determine whether age influenced the course of glycolysis. It was found that the capacity of an aqueous extract of lens to reduce hexose diphosphate to triose diminished markedly with the age of the lens. It was also found that the equilibrium reaction for equation hexosediphosphoric acid $\longleftrightarrow$ triosephosporic acid became impaired with age.

F. H. ADLER.

Methods of Examination

A TRIAL SPECTACLE FOR PRISM PRESCRIPTION. I. C. MICHAELSON, Brit. J. Ophth. **21:** 232 (May) 1937.

The purpose of the trial frame described is to determine in a practical way the amount of phoria to be neutralized. The frame is light and can be borne without discomfort when worn with the usual spectacles. Each cell contains a 4 prism diopter lens. Both prisms can be simultaneously rotated by a screw so as to give a maximum of 8 prism diopters. The patient is given the spectacles for home use and is directed

how to turn the screw which rotates the prisms. He is told to increase the degree of prism about 1 D. at a time until he has reached a point where maximum comfort seems to have been obtained. This is repeated for several evenings. The surgeon then has definite evidence on the basis of which to prescribe prisms and knows not to do so should the patient have obtained no relief by their trial use. There is one illustration.

W. ZENTMAYER.

Neurology

HERPES ZOSTER OPHTHALMICUS AND OPHTHALMOPLEGIA. J. VOISIN, Bull. Soc. d'opht. de Paris, June 1936, p. 399.

Voisin reports a case of herpes zoster ophthalmicus and ophthalmoplegia in a woman 73 years of age in which the third, fifth and sixth cranial nerves were involved. The syndrome originated with herpes of the left eye accompanied by ptosis. In addition to loss of sensibility of the cornea, the eye was proptosed, and movement was limited in all directions of gaze. The pupil was in a state of mydriasis and did not react. The corneal epithelium showed irregularities, and there were a few signs of intra-ocular inflammation. Visual acuity was lowered to 1/20 with correction. General physical examination resulted in no positive findings. In two months no appreciable recovery was noted. Contributions to the literature concerning patients with a similar condition by Rebattu, Monnier, Kühn, Dechaume, Bonnet and Colrat, Valière-Valeix, and Veil and Isnel are cited. Voisin believes the infection may be carried into the cavernous sinus, causing basilar meningitis.

L. L. MAYER.

CORNEAL LESIONS AFTER REMOVAL OF THE GASSERIAN GANGLION FOR TRIGEMINAL NEURALGIA. C. DRUTTER, Deutsche Ztschr. f. Chir. **248:** 55 (Dec. 9) 1936.

Drutter reports that among sixty-nine patients treated for trigeminal neuritis by the injection of alcohol into the gasserian ganglion, keratitis developed in seventeen (24.6 per cent). In nine (52.9 per cent) the lesion became permanent. Eighteen patients were subjected to twenty-seven operations for removal of the gasserian ganglion. The cornea remained normal in all cases. Drutter concludes that neuroparalytic keratitis develops only as a result of anesthesia of the cornea. The cause of keratitis remains unexplained in spite of many studies. Primary trigeminal neuralgia seldom involves the first branch of the nerve. Pain in the area of this branch is considered secondary by many authors. The incidence of corneal anesthesia and consequent disease of the cornea cannot be diminished through the use of the injection of alcohol into the gasserian ganglion as developed in Germany. Subtotal posterior ganglion section of the root of the trigeminal nerve performed according to the method of Frazier and Spiller does not lead to loss of sensitiveness of the cornea and to neuroparalytic keratitis. The persistence of a high mortality in Germany following the operative intervention is due to lack of experience. Treatment with the injection of alcohol makes the operative intervention even more difficult. Subtotal section is followed by a smaller percentage of

recurrences than the injection of alcohol. According to American authors, subtotal resection of the posterior ganglion is the method of choice in the treatment of trigeminal neuralgia. Drutter suggests that the method of injection should be reserved for patients whose general condition does not justify a major operative procedure. Younger patients should be treated, as far as possible, by operative intervention so as to avoid the possibility of keratitis.

J. A. M. A. (W. ZENTMAYER).

GENICULATE BODIES AND OPTIC CORTEX IN ATROPHY OF THE OPTIC NERVE OF ONE ENUCLEATED EYE. N. VON HORÁNSKY, Klin. Monatsbl. f. Augenh. **97:** 438 (Oct.) 1936.

A man aged 61 had lost his left eye eighteen to twenty-two years before. The optic nerves, chiasm and optic tracts were histologically examined after the patient died from cancer of the colon. A detailed description is given of the sections stained after the method of Weigert-Pal and of Nissl. The observations in this case of peripheral blindness supported fully the view of Minkowski regarding the crossed and uncrossed fibers of the optic nerve in the geniculate bodies. The calcarine cortex on each side was found intact. The examination of the calcarine cortex did not corroborate the view of Kleist, Schröder and Henschen regarding the homolateral and contralateral termination of the visual tracts. The results obtained in this case seem to indicate that the brains of one-eyed patients cannot be used to solve the latter question, at least by the present methods.

K. L. STOLL.

Ocular Muscles

THE STEREOSCOPE IN THEORY AND PRACTICE, ALSO A NEW PRECISION TYPE STEREOSCOPE. E. KRIMSKY, Brit. J. Ophth. **21:** 161 (April) 1937.

The considerations in the paper are grouped under two main headings—theoretical and practical.

Among the author's conclusions are the following:

Testing with a fixed stereogram (with the exception of a phoria card) is an unsatisfactory method of determining the status of fusion or for orthoptic training.

Testing with a split stereogram which can be shifted to correspond to changing positions of the visual axes with variable accommodations is the only practical method for stereoscopic study.

The ordinary professional stereoscope, because of its cramped construction and lenses of fixed focal length, is considerably reduced in its adaptability to excessive convergence and divergence. The stereoscope, however, may be so modified as to be adaptable to fairly large degrees of ocular convergence by the use of widely separated lenses of selective short focal lengths and supplemented, if necessary, by suitable prisms.

The incorporation of fixed base-out prisms, as in the ordinary stereoscope, is not, as is ordinarily supposed, a means of adapting the instrument to greater convergences but is a relic of bygone days for

employing larger stereograms (a greater field of view) for purposes of parlor entertainment. The addition of selective prisms should be left to the examiner, who alone is in a position to determine the need for supplementing base-in or base-out prisms.

The author's stereoscope enables the examiner to determine at a glance the amount of convergence and divergence, with selective accommodations, necessary for focusing split pictures, by incorporation of: (*a*) viewing lenses of variable separation and of known focal length, (*b*) a movable calibrated rod, (*c*) viewing boxes calibrated to record the amount of separation of split pictures and (*d*) tables to which the examiner may refer so as readily to translate these readings for vergence.

W. ZENTMAYER.

Orbit, Eyeball and Accessory Sinuses

TWO CASES OF INTERMITTENT EXOPHTHALMOS; VARICOCELE OF THE ORBIT. F. HIPPERT, Arch. d'opht. **53:** 135 (Feb.) 1936.

Up to the time of this report ninety-one cases of intermittent exophthalmos have been described. Hippert reports two new cases, making a total of four which have been seen in Weill's clinic at Strasbourg. These two cases are fully described and are illustrated by photographs clearly showing the condition. The characteristic features are then briefly reviewed. The condition remains stationary for years, the most serious complication being hemorrhage into the orbit and into the brain. The prognosis for life is good, but that for vision is less favorable. Few patients have recovered spontaneously, and in only four cases has a cure been effected. The author favors sclerosing injections because of the great surgical risk. In neither of the two cases reported was treatment curative. Vestibular symptoms were present in one case, suggesting the possibility of vestibular varices.

S. B. MARLOW.

OCULOHYPOPHYSIAL SYNDROME FOLLOWING SUPPURATIVE SPHENOID SINUSITIS; ANATOMIC-PATHOLOGIC EXAMINATION. G. WORMS, Arch. d'opht. **53:** 207 (March) 1936.

Worms presents in detail the history, course and results of postmortem examination in a case of sphenoid sinusitis of long standing which had produced visual disturbances and general physical changes constituting the essential elements of the adiposogenital syndrome. The history is noteworthy because of the grouping and predominance of symptoms which ordinarily would necessitate the diagnosis of tumor, although no tumor was found. The atrophy of the optic nerve was shown by histologic study to be the result of optic neuritis of sphenoid origin. A hypertrophic reaction of the hypophysis was also demonstrated to be the result of inflammation. The sphenoid sinusitis was completely overlooked, there being no symptoms pointing to it. The facts in the case studied demonstrate clearly that the meninges at the base of the brain and about the optic nerve can be involved in the reaction to an inflammation in the sinuses, perisinusitis.

S. B. MARLOW.

EXOPHTHALMOS DUE TO A DILATING PNEUMOSINUS OF THE ETHMOID. P. BONNET, Bull. Soc. d'opht. de Paris, February 1936, p. 109.

The term "pneumo-sinus dilatans" is reserved for a condition distinguished from pneumatocele in that a single cell of a sinus becomes filled with air and gradually increases in size. No mucocele accompanies this condition, and no liquid is found in the cell. In a thesis on this subject by Benjamins, presented at Lyon in 1935, ten cases were recorded. The present report concerns a boy 15 years old who presented himself to the clinic with exophthalmos of the left eye that had followed a severe blow to the left superciliary region. No epistaxis or ecchymosis occurred, but the proptosis was painful. It did not increase. The protrusion was 4 mm. anterior to the cornea of the fellow eye. Visual acuity was reduced to 1/8. The results of rhinologic examination, together with findings shown by a roentgenogram, warranted the diagnosis of ethmoid mucocele. The results of general physical examination were of no importance. However, at operation only a large ethmoid cell crowding the orbital contents was found. When opened this contained only air. Exophthalmos regressed fairly rapidly, and about two weeks later the exophthalmometer reading for each eye was 14 mm. There was no increase in visual acuity in the damaged eye.

L. L. MAYER.

Physiology

THE PRECORNEAL LIQUID LAYER. J. ROLLET, Arch. d'opht. **53**: 5 (Jan); 111 (Feb.); 255 (April) 1936.

Rollet discusses in detail the superficial moist covering of the cornea from the standpoint of its physiologic and pathologic features, the etiologic variety of the syndromes of the preocular cavity and the therapeutic deductions which can be made. Among the various conditions that have some relationship to the fluid covering of the cornea are included neuroparalytic keratitis, ulcer of the cornea and conjunctivitis, the conjunctival and palpebral symptoms of sleep, severe general disease, the oculonasal catarrh of measles, ophthalmic zoster, intermittent pain following a slight corneal injury, filamentary keratitis, corneal lesions due to avitaminosis, corneal dystrophies and a number of other corneal disturbances. The author believes that not enough attention has been paid to the functions of this fluid, which is preocular, and the glands which produce it. It is composed not only of salts and water derived from the lacrimal glands but has properties of viscosity and adherence due to the content of mucin derived from the glands of the lids and conjunctiva. It plays an important part in the dioptric function of the cornea. A second function is the mechanical protection it affords. Most important of all, it is indispensable to the normal trophicity of the superficial epithelium. This trophic rôle must be considered from the point of view of its nervous mechanism, motor (lids), sensory (cornea) and secretory sympathetic. Alteration or deficiency in this fluid can result in anatomic lesions of the superficial corneal epithelium, accompanied by functional symptoms of which blepharospasm, photo-

phobia and tearing constitute a triad. Frequently a disturbance of the sympathetic nervous system must be suspected. In dealing with conditions affecting the epithelium much more attention should be paid to the quality of this fluid than to its quantity. In order not to disturb its formation and in case of failure of reestablishment of its secretion in integrity, one should be able to make up for its insufficiency indirectly (by a bandage, a contact glass or blepharorrhaphy) or to use a collurium which not only is bactericidal but is also eutrophic for the subjacent cells, covering them for the longest possible time owing to properties of adherence and viscosity, without the necessity of too frequent instillations.

S. B. MARLOW.

Refraction and Accommodation

HIGH MYOPIA WITH GOOD VISION. E. TRON, Sovet. vestnik oftal. **10:** 19, 1937.

Tron examined two groups of eyes: (1) eyes with high myopia with poor vision and (2) eyes with high myopia with good vision. The refraction was determined with the eye under the influence of atropine; the radius of the corneal curvature was examined with the ophthalmometer; the depth of the anterior chamber and the thickness and the radii of the curvature of lenticular surfaces were measured with Tscherning's ophthalmophakometer. The refraction of the cornea and the lens and the length of the axis of the eyball were also measured. Tron reached the following conclusions:

1. The ophthalmophakometric measurements showed that axial myopia was equally prevalent in both groups of eyes.

2. The length of the axis is not the deciding factor in good vision; in some patients with high myopia with good vision the axis was of considerable length (from 29.65 to 31.8 mm.), while in a number of patients with poor vision the axis was not lengthened.

3. A few patients with high myopia with good vision had changes in the macular region, while there were no such changes in some patients with poor vision. Therefore the changes in the macula are not the only factor influencing visual acuity.

4. The refractive power of the eye is one of the important factors influencing visual acuity. The ophthalmophakometric measurements showed that in persons with high myopia with good vision the refractive power of the eye is considerably less than in persons with high myopia with poor vision, chiefly because of the low refractive power of the lens. This leads to increase of the size of the retinal image, i. e., to better visual acuity.

5. The most important factor is the unequal value of the cones in various eyes (illustrating Hecht's theory of the increase in number of the functioning cones with better illumination); most likely there is a variation of the cones in relation to their cross-section.

O. SITCHEVSKA.

Retina and Optic Nerve

SURGICAL TREATMENT OF SEPARATED RETINA BY THE GALVANIC METHOD. C. B. WALKER, Am. J. Ophth. **19:** 558 (July) 1936.

This article does not lend itself to abstracting. Walker arrives at the following conclusions:

"1. An attempt to evaluate the possibilities of closing retinal tears by electrolytic alkali, by catholysis as described by Vogt, has confirmed his statements in practically every detail after 15 operations modified from Vogt.

"2. Every type of case (except the case illustrated by figure 6) described by Vogt has been observed, and three separated retinas, perhaps more extensively damaged than any included in the above report, are briefly recorded here.

"3. The upper limit of alkali treatment by catholysis was not proved, although it was believed that a record was set when 126 micro-needle cathode punctures of about one second terminal duration, carrying an average of .85 M.A. of current on a .006"-diameter needle penetrating 7 mm., were used with every evidence of success. Also 1/50 cc. of 3-percent KOH was injected subchoroidally at the macular region in the same eye. (Recently 168 micro-needle punctures for tears involving over half of the eye, were well tolerated, and resulted in improvement but not a cure.)

"4. However, failure occurred in a case with a traumatically torn hole in the retina, from two to three times the size cited by Vogt, extending centrally 45 degrees from the macula. But in two cases a large 70- to 75-degree dialysis below, also with a macular hole, was reattached, with excellent field and vision, both in a myopic eye and in a case of traumatism.

"5. Indications point to the fact that perforation of the retina is not necessary in all cases, although it is often unavoidable by micro-needle galvanic alkali treatment (catholysis).

"6. An extremely fine (.003") micro-needle supported, or trocar type of 25-percent iridium-platinum, has important characteristics as regards inhibiting leakage and bleeding.

"7. Detachable micro-needles may be used to treat more posterior areas, but they are especially valuable to relocalize in case of deceptive tracer bubbles and scleral coördination."

W. S. REESE.

MASSIVE RETINAL FIBROSIS IN CHILDREN. A. B. REESE, Am. J. Ophth. **19:** 576 (July) 1936.

Reese encountered in young persons several conditions characterized by the protrusion of a grayish white mass from the retina due to organization of hemorrhage occurring at birth or resulting from trauma after birth. He reports three cases of this condition, giving the clinical and pathologic findings, and states the following conclusion:

"Retinal hemorrhage in children can organize into a fibrous mass resembling clinically a retinoblastoma. Due to contracture, more and more of the retina can be pulled into the lesion, giving it the appearance

of progressing, and thereby leading all the more to the suspicion of a retinoblastoma. There is usually no detachment of the retina because the lesion is adherent to the choroid. The retinal hemorrhage occurs at birth from the same conditions that produce the analogous condition of massive intracranial hemorrhage. It may occur as the result of trauma in early childhood."

W. S. REESE.

CYSTS AND EPENDYMOMAS OF THE RETINA. C. DEJEAN, Arch. d'opht. 53: 81 (Feb.) 1936.

Dejean believes that the application of the principles of the general pathology of the central nervous system if applied to retinal pathology can go a long way toward clarifying disputed and obscure points. He refers briefly to the work he has already done over a period of years and calls special attention to the researches of Redslob, with whom he is in general agreement. In this report he first discusses cystic malformations of the retina, calling attention to their embryologic relations with ependyma. Cysts of the iris are included, as they are formed between the two pigmented posterior epithelial layers of the iris. He suggests that detachment of the retina can be looked on as a retinal cyst and is a reconstitution of the intra-ocular ependymal cavity by the collection of fluid from various causes. Tubular formations of the ciliary body are similarly discussed, and the conclusion is drawn that they are regressive metaplasias and that true adenoma of the ciliary body does not occur for the reason that this part does not have a secretory function. In a section on tumors of the infantile retina the author expresses the opinion that the term glioma should be rejected. If the cells resemble those of the primary epithelium of the ependymal cavity the tumor should be considered a neuro-epithelioma; if they are like those of the neurospongioma surrounding the canal the tumor should be considered a neurospongioma, and if they are like the cells already differentiated into rentinal neurons, the tumor should be considered a retinocytoma. Examples, illustrated by photomicrographs, are described. In the last section tumors of the adult retina are considered. These include pigmented epithelioma of the iris and primary ciliary and cilioretinal epithelioma. Many of these conditions can be explained on the basis of the relations of normal and pathologic homologies between the retina and the ependymal walls, or the cerebral ventricles.

S. B. MARLOW.

PRELIMINARY NOTE ON THE INNERVATION OF THE OPTIC NERVE IN MAN. J. MAWAS, Bull. Soc. d'opht. de Paris, February 1936, p. 87.

In the course of research on the vegetative nervous system Mawas made some observations concerning the optic nerve which have not previously been described. A rich nerve plexus was found surrounding the optic nerve. The name nervi nervorum opticorum is proposed for this group of fibers. An outline of the special technic necessary for the histologic study of these nerve elements is given. The author is unable to say at the present time what the exact destination of the fibers is, but it is probable that they subserve the membranes and vessels in

the vicinity. Two excellent photomicrographs accompany the article. Mawas believes that further study by him will answer the question of the function and destination of these nervi nervorum opticorum.

L. L. MAYER.

THE REFLEX STREAKS ON THE RETINAL VESSELS. ALFRED JAEGER, Arch. f. Augenh. **110:** 137, 1936.

Being a physiologist and not an ophthalmologist, Jaeger has studied the light streaks on the blood vessels in the eye by comparing them with similar streaks on the mesenteric vessels of lower animals. His chief interest has been to show that the moving column of blood in the vessels is one of the chief factors in producing the bright light streak in the arteries. He believes that the central portions of the column of blood which are moving more swiftly tend to orient the blood cells in such a way that they all lie with their broad surfaces in the long axis of the vessel, and this makes for a more perfect reflecting surface than the cells which line the walls of the vessels. These cells, moving slowly, are apt to have their edges turned toward the observer, or irregularly placed so that they do not reflect the light as well.

Jaeger concludes that the general background of the vessels is important and that the walls of the vessels, although translucent, cast a shadow which sets off the central light streak. In the frog, for example, which shows a relatively dark background, the central light streak is not nearly as bright as the two reflections from the walls of the vessels. In man and in the dog there are two kinds of reflex streaks: 1. A small bright bluish white streak which comes from the membrana limitans interna of the anterior wall of the vessel. This is often broken corresponding to the curvature of the vessel and is found in man on the veins as well as on the arteries. 2. A broad yellowish red streak which is reflected through the wall of the vessel from the retina and which is reflected back to the observer from the column of blood because of its properties, which have already been mentioned. It is found only in the arteries in man and is absent in these when the column of blood has stopped moving.

These two light streaks can be differentiated from each other by moving the light of the ophthalmoscope on the vessel under observation. The first one follows the movement of the source of light clearly, while the latter remains stationary.

F. H. ADLER.

ORIGIN AND SIGNIFICANCE OF GUNN'S SIGN. E. HORNIKER, Klin. Monatsbl. f. Augenh. **97:** 315 (Sept.) 1936.

Horniker reviews the publications on Gunn's sign, beginning with this author's paper. A complete bibliography on the subject and on related questions is appended. Referring to the research of Salus and to that done jointly by Sallmann and Kahler, Horniker describes in detail modifications in the use of palpatory sphygmomanometry by means of Boulitte's apparatus and the manner in which he employs oscillometry after the method of Plesch. After describing these methods

and other oscillatory methods adduced for comparison, Horniker states his own views on the vascular condition in patients who present Gunn's sign and Salus' arc. He points out that pulsatory phenomena in the retinal vessels may be observed even in healthy persons not only with the large ophthalmoscopes but also with the small electric ophthalmoscope. Jonas Friedenwald's terminology of the pulsations is discussed and commented on favorably.

With reference to the origin of Gunn's sign, Horniker distinguishes two phases. Atony of the central retinal artery, which possesses only a few muscle fibers, precedes arteriosclerosis and is of great importance during the first phase. The first pathologic changes are produced by increased expansive pulsation in the wall of the artery.

The second phase is the hypertony that occurs with advanced age; it narrows the bore of the artery by increased tonus. Then the walls of the veins, which were compressed by preceding changes, become visible at a certain distance from, and on both sides of, the artery, thus producing Salus' arc. Hence the initial stage of arteriosclerosis plays a predominant rôle in the genesis of Gunn's sign. Discussing the changing location of the retinal vessels which takes place during the course of observation, as described by Westkamp, Horniker suggests that photographs be taken systematically for several years. Simultaneously, oscillograms and ophthalmodynamometric data should be recorded.

K. L. STOLL.

Trachoma

TRACHOMA. R. E. WRIGHT, Brit. J. Ophth. **21:** 198 (April) 1937.

Wright briefly reviews the results obtained from experiments based on the observation of the appearance of growths on the chorio-allantoic membrane of the chick when inoculated with unfiltered trachoma material. The laboratory aspect of the work was side-tracked since controls revealed that a variety of agents other than known viruses and unfiltered trachoma material produced similar growths on the chorio-allantoic membrane with varying histologic changes. It may be that these findings put an end to the original idea, but until the whole question of the effects of different agents (organismal, chemical, mechanical known virus, etc.) on the chorio-allantoic membrane is worked out, nothing further can be said as to the significance of the effect of inoculation with unfiltered, but apparently bacteriologically sterile, trachoma material.

W. ZENTMAYER.

TRACHOMA AS A VIRUS DISEASE. A. F. MACCALLAN, Rev. internat. du trachome **14:** 5, 1937.

Inclusion bodies, noted in cases of trachoma in 1907, have been since found in all virus diseases, but only in such diseases. Cellular inclusions have two components: (1) gram-negative elementary bodies, Chlamydozoa, consisting of dustlike granules from 200 to 300 millimicrons in diameter, embedded in a ground substance capping the nucleus and (2) larger initial bodies, about the size of a minute coccus, which may or may not be free. Both bodies are considered different forms of the

same organism. The prevention of trachoma lies in the avoidance of contagion, and the disease is probably contagious until the late phase of stage III is reached.

J. E. LEBENSOHN.

PATHOLOGIC CHANGES IN LATE TRACHOMA. A. BIRCH-HIRSCHFELD, Rev. internat. du trachome **14:** 13, 1937.

Characteristic of advanced trachoma is an excessive neoformation of fibroblasts occurring first in the subepithelial and pretarsal tissue and later involving the tarsus. Since the proliferation of fibroblasts is the sequel of the process of infiltration by lymphocytes and plasma cells, the serious sequelae of trachoma are best avoided by early energetic treatment.

J. E. LEBENSOHN.

TRACHOMA AND BACTERIUM GRANULOSIS OF NOGUCHI: COMPLEMENT FIXATION TEST IN TRACHOMA. E. TRAPEZONTZEVA, Sovet. vestnik oftal. **10:**101, 1937.

The complement fixation test was done with the immune serum of rabbits and with serums of patients suffering from trachoma. The culture of Noguchi's bacillius was used as an antigen; its antigenic and agglutinable character was studied previously in view of its anti-complementary properties. The antigen was prepared in various ways: by irradiation with ultraviolet rays, by heating and by treatment with solution of disodium sulfate. It was carefully titrated. The serum of healthy persons served as a control.

Two hundred trachomatous patients were examined. The complement fixation test was positive in 57 per cent. These included patients with progressive trachoma and those incompletely cured. The complement fixation test was negative in doubtful cases or in those cases in which cure was temporary or permanent. The reaction of the control serum was also negative. The reaction was positive in the following percentages in cases classified according to the various stages of trachoma: trachoma of stage 1, 34.5 per cent; trachoma of stage 2, 75 per cent, and trachoma of stage 3, 51.8 per cent. In order to obtain accurate data the serum had to be fresh and inactivated not longer than one or two days previous to the test. The reaction should be read as soon as hemolysis is discovered in the test tubes containing the control.

O. SITCHEVSKA.

Tumors

LYMPHOSARCOMA OF EYELID. V. M. MÉTIVIER, Brit. J. Ophth. **21:** 202 (April) 1937.

Sarcoma of the eyelid is a rare growth, but Smith and Elmes in an analysis of five hundred tumors among the natives of Nigeria found ten cases of round cell sarcoma of the orbit. These authors suggested that probably many of these growths had their origin in trauma, since injuries to the eyes are notably common among natives.

Métivier reports a case of lymphosarcoma of the eyelid. The patient was a brown-skinned boy of impure African descent living in Trinidad.

When he was first seen the condition appeared to be a large untreated chalazion. Antisyphilitic treatment was given, as the Wassermann reaction was strongly positive. No effect was noted from the treatment, and as the growth at that time completely hid the globe and there was an extension toward the subconjunctival tissue of the lower lid, with invasion of the caruncle and plica, exenteration of the orbit, with removal of the eyelids, was done. Histologic examination proved the growth to be a large round cell sarcoma. An enlarged lymph node on the angle of the jaw showed the same structure. Two years later there was no evidence of recurrence and the patient was in good health.

This article is illustrated.

W. ZENTMAYER.

SEBACEOUS TUMORS OF THE LIDS. G. MORARD, Bull. Soc. d'opht. de Paris, June 1936, p. 435.

Morard reviews the literature on tumors developing in the sebaceous glands of the lids. The embryologic aspects of these glands as discussed and pictured by Ida Mann is commented on. On the basis of an anatomic study of the meibomian and ciliosebaceous glands, the cellular elements are divided into four groups—generative cells, transitional cells, sebaceous cells and reticular formation.

Morard groups the cutaneous sebaceous tumors into sebaceous adenoma (typical and metatypical) and sebaceous epithelioma (basosebaceous, spinosebaceous and mixed metatypical and sebaceous).

The meibomian tumors are classified into adenoma and epithelioma.

Reports of cases are included, illustrated with excellent photographs showing the clinical and microscopic features. The conclusions are that the embryologic and histologic analogies between the meibomian glands and the cutaneous sebaceous glands establish a parallel between the neoplasms developing along these two orders of formation. Therapeutically, the sebaceous meibomian epitheliomas are markedly radiosensitive as compared with the cutaneous sebaceous epitheliomas. An excellent bibliography is appended.

L. L. MAYER.

Therapeutics

EFFECT ON THE EYE OF RADIUM USED FOR TREATMENT OF MALIGNANT DISEASE IN THE NEIGHBORHOOD. PHILIPPA MARTIN, Brit. M. J. 1: 651 (March 27) 1937.

Martin has studied the effect of radium on the eye in cases of tumor of the skin and of the upper jaw in which this treatment was used. The treatment consisted in part of interstitial irradiation with needles of low linear intensity or with a 1 Gm. radium unit. Needles should have a screen of 0.8 mm. of platinum in all cases in which it is necessary to introduce radium into the orbit. The reactions appeared in the conjunctiva either early or late; congestion of the iris occurred, which was never serious, and necrosis of the cornea was seen, which was the most serious result and was usually associated with radium necrosis of skin or bone. The necrosis of the cornea bears some resemblance to neuroparalytic keratitis; it rarely takes place rapidly. If the cornea becomes affected it should be protected by a suture of the eyelids.

Radium cataract is usually a late reaction, occurring two or more years after exposure. Martin mentions an unusual case of early mature radium cataract. To protect the eye when the surrounding tissues are to be irradiated it is best to stitch the lids together. The lids should be split and the sutures left in place for four weeks. The use of the 1 Gm. unit produces so little reaction that preventive treatment is not necessary. It is well to keep the eye under observation in a case in which it has been subjected to intense irradiation, as necrosis of the cornea generally comes on late, namely, about eight weeks after exposure.

The author concludes as follows:

"The observation of eyes which have been exposed to radium irradiation of destructive intensity has demonstrated a series of morbid changes leading progressively to radium necrosis of the cornea and loss of the eye

"When treatment of neighbouring malignant tumors is by interstitial irradiation these changes can be reduced by adequate screenage of the radium needles.

"After mass irradiation by a large quantity of radium at a distance the damage to the eye is negligible.

"As greater quantities of radium become available, and facilities for the use of mass irradiation become more widely distributed, it is to be hoped that damage to the eye by interstitial irradiation will cease to be of more than historic interest."

A. KNAPP.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

Forty-First Annual Meeting, New York, Sept. 26 to Oct. 3, 1936

FRANK E. BURCH, M.D., St. Paul, *President*

WILLIAM P. WHERRY, M.D., Omaha, *Secretary*

CLINICAL PROBLEM OF ALLERGY IN RELATION TO CONJUNCTIVITIS AND IRITIS. DR. ALAN WOODS, Baltimore.

This article was published in full in the January 1937 issue of the ARCHIVES, page 1.

PRIMARY SARCOMA OF THE UVEAL TRACT: AN ANALYSIS OF TWENTY-SEVEN CASES. DR. S. HANFORD MCKEE, Montreal, Canada.

In the uveal tract, sarcoma appears much oftener in the choroid than in the iris or the ciliary body. E. Fuchs (quoted by Greeff: Pathological Anatomy, Berlin, A. Hirshwald, 1903, p. 296) estimated that in 6 per cent of the cases of sarcoma of the uveal tract the iris was affected; in 9 per cent, the ciliary body, and in 85 per cent, the choroid.

Malignant melanoma is relatively rare. According to Fuchs (Das Sarcom des Uvealtractus, Vienna, W. Braumüller, 1882), it occurs in about 0.066 per cent of all cases of ocular conditions. Stallard (in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 689) stated that during 1925-1931 at Moorfields sarcoma of the choroid was found in 1 of every 4,000 patients attending the clinic for outpatients. It occurs mostly between the fortieth and sixtieth years, though in rare instances it has been found in children. Males and females are affected about equally. Rarely does this disease affect both eyes.

The origin of the essential pigment of the melanin of the eye may have an important pathologic bearing on the nature of intra-ocular tumors: Bloch isolated from the embryo of the broad bean 3, 4- dihydroxyphenylalanine, a substance which he called dopa, and showed that it was readily changed by this oxidase to melanin. When this substance is added to epidermal cells of skin in frozen sections fixed in formaldehyde, granules of melanin are formed (the dopa reaction).

Melanin is closely allied to epinephrine, and it is probable that the two substances are derived from the same precursors and form alternative end-products in metabolism. When the cells are stimulated to proliferate so that there is an increase of the oxidative ferment, a pigmented tumor is the result, or generalized melanomatosis may follow, as in Addison's disease. It would seem from clinical observations that in

the eye melanogenesis is a function of both epiblastic and mesoblastic tissues, and recent chemical work seems to substantiate this deduction. The chemical findings fall in line with the clinical observations, and, so far as the eye is concerned, it seems clear that pigmentation may be either ectodermal or mesodermal, so that malignant melanoma may be either carcinomatous or sarcomatous. In respect to this Terry and Johns (*Am. J. Ophth.* **18:** 903, 1935) stated: "In recent years grave doubts have arisen as to the mesoblastic origin of the class of primary malignant uveal neoplasms commonly called sarcomata. In fact, Masson and others have presented strong evidence that is indicative of ectodermal origin."

In 1931 Major Callender (*Tr. Am. Acad. Ophth.,* 1931, p. 131) described four specific types into which all primary malignant uveal neoplasms can be classified:

1. *The Spindle Cell Type.*—The characteristics of this type of tumor are sheets, whorls and other irregular arrangements of spindle-shaped cells with long oval nuclei. The ends of the cell appear to terminate in fibers, so that the cells resemble fibroblasts. There is no argentophil reticulum except that accompanying the nutrient vessels. These spindle cell tumors can be separated into the following two divisions by the nuclear characteristics.

Subtype A: The nucleus has a delicate reticular structure in which the nucleolar material is not well defined. These tumors are usually fairly heavily pigmented.

Subtype B: The cell has a sharply defined, deeply stained, small round nucleolus, usually situated near the center of the nucleus in a rather coarse nuclear network. These tumors are usually lightly pigmented.

2. *The Fascicular Type.*—The cells of this type of tumor are elongated, sometimes forming fiber-like structures, but rounded and polygonal shapes are seen. The characteristic nucleus is oval, has a distinct nucleolus and closely resembles the nucleus of the cell of the spindle cell tumor of subtype B. The majority of the cells are arranged in columns or fasciculi, the long axis of the cell being at right angles to that of the column. The cells radiate about the center of the column in a palisade arrangement, the center being a lymphatic or a capillary blood vessel. Pigmentation is usually scanty.

3. *The Epithelioid Type.*—Tumors of this type have polygonal cells, usually of relatively large size, though there is considerable variation in both the size and the shape. The nucleus is large and round or somewhat oval, and the nucleolus is distinct. This type varies greatly in the degree of pigmentation.

4. *The Mixed Cell Type.*—Tumors of this type have irregular mixtures of cells of the spindle and epithelioid types, with occasional areas of cells of the fascicular type. These tumors are heavily pigmented.

A recurrent orbital tumor has been noted rarely, and in this series was present in only 1 case. Terry and Johns suggested that the orbit may be an unfertile field for tumors and that Tenon's capsule may hold the tumor in check at least long enough for a metastasis already existing or a metastasis from the recurrent orbital growth to bring about a fatal issue before local recurrence manifests itself.

The liver is the most fertile field for a metastatic growth, and while most metastatic tumor cells are blood borne, metastasis by way of the cervical glands does occur.

DISCUSSION

DR. T. L. TERRY, Boston: Primary tumors of the iris appear to be less malignant than those of the choroid, and they should be regarded as in a different category.

I wish to disagree with the essayist and the many others who insist on dividing tumors of the uveal tract into the four stages. Such a division is not only artificial but also extremely misleading. The term "stages" immediately brings to mind a picture of development and metamorphosis in a sequential order. It also tends to lend a false sense of security if the eye is enucleated when the tumor is small, the retina unseparated, glaucoma not present and the eye unruptured. In the series reported by Johns and myself in 1935, stage four (metastasis) was reached in 1 instance even before the complete development of stage one, i. e., before separation of the retina. Stage four (metastasis) was reached before stage two (glaucoma) in 7 instances. And, further, stage four (metastasis) was reached before the advent of stage three (extension of the tumor outside of the globe) in 17 instances. It must be stressed that metastasis may occur early, even before retinal separation. One is never sure of cure by enucleation. The prognosis cannot be judged by either the size or the so-called stage of the tumor. Therefore, it is never safe to delay enucleation after the diagnosis is made.

The most striking feature of Callender's classification according to cellular type is that not in a single case of tumor of the spindle cell subtype A did death occur. Is this type a benign melanoma of the choroid, capable of destroying the eye and even invading the orbit? The differentiation of the other types may eventually give a fairly accurate method of grading malignancy. A study of the reticulum as stained by the silver stain of Wilder is of value. The value of the Klein test as applied by von Hippel to malignant melanoma of the uveal tract must still be proved.

When one analyzes the paper of the essayist one finds much of practical clinical information. It shows the importance of the following points, which I stress at every opportunity.

1. One should suspect malignant melanoma in every case of separated retina.

2. One should suspect malignant melanoma in every case of unilateral glaucoma.

3. One should suspect malignant melanoma in every case of unilateral uveitis. In tumors of this type, when there is a great deal of necrosis or even a moderate amount, uveitis is likely to occur. Of course, it is unilateral.

4. One should suspect malignant melanoma in every blind eye, especially when it is impossible to see the fundus. A number of ophthalmologists advocate operating on a blind eye with glaucoma in order to reduce the pain and make the eye comfortable, but in our experience of 97 cases a tumor was found after the eye was opened in some 40 instances, and it was not even suspected clinically. I have talked

about this so much at our local institution that the house officers now record for almost every eye that is removed a note of "possible sarcoma." That may be something of a joke, but at least it is in their minds for clinical differentiation at the time.

5. Finally, it should be remembered that an intra-ocular operation may stimulate the tumor to grow more rapidly and become more malignant, as well as furnish a tract of decreased resistance through which it may grow. In fact, some observers have stated that the most common channel by which the tumor extends from the eye is along the tract of an operative wound.

Sir John Herbert Parsons, London, England: The ordinary and flat sarcomas, I think, are somewhat like the secondary carcinomas which grow in between the planes of choroid tissue. I think they are located among the lymph spaces of the choroid, and this points to the origin of the flat sarcomas, which also have a sort of alveolar structure: In all probability, the flat sarcomas, which are rare, are endotheliomas growing in the tissue spaces.

With regard to malignancy, there is, first, the question of pigmentation to be considered. I think that pigmentation has little reference to malignancy in cases of sarcoma of the choroid. Most of these tumors are pigmented, but one does see malignant true leukosarcoma. Some of the pigmented tumors are really leukomas which have hematogenous pigmentation. They are probably usually of the angioid type, not in the ordinary sense of the word but in the sense that they are of the vascular type of sarcoma. In tumors of this type the pigmentation is of a different kind. Even in such tumors the hematogenous pigmentation may become iron-free, and it would be impossible to distinguish it from the other type of pigmentation. I do not believe the pigmentation has much to do with the malignancy, but the fact that the growth is pigmented makes one extremely suspicious of malignancy, unduly so. That is due to the fact that a vast majority of tumors are pigmented.

The most important point about a malignant growth is the site of the tumor in the eye. If it is discovered when it is at an early stage, the eye can be enucleated without delay. I think, from my experience, that in these cases the prognosis is good with regard to subsequent developments. But if the tumor is situated at the periphery it is less likely to be detected as early as a central growth. Consequently there is delay, which is often considerable, before the eye is removed. and the prognosis is much worse than in the case of a central tumor.

Clinical Importance of Pigment in the Fundus. Dr. Arthur J. Bedell, Albany, N. Y.

Variations in the pigmentation of the fundus assist in the diagnosis of ocular diseases and are often a reflection of the condition of the general health. The color of the fundus depends on the amount of pigment in the hair and skin. Congenital deposits of pigment are of no pathologic significance but may confuse the uninitiated. They are flat collections of pigment situated near the disk and peripherally. Senile pigmentation is an exaggeration of drusen, Tay's choroiditis and senile macular degeneration. The benign drusen may become so large and so central as to confuse the ophthalmologist who does not use the dis-

criminating tests for vision and defects of the field. Inflammation of the retina is always accompanied by migration of the pigment. The picture depends on the number and size of the patch or patches. Many characteristic forms have been described during the eighty years of ophthalmoscopy. When the retina is detached, the appearance of unusual pigment may be the determining factor in diagnosing an underlying malignant growth. All penetrating wounds of the sclera and all severe contusions of the globe leave pigmentary evidence of damage. The degree and site of the lesion are often of great medicolegal value. Of particular importance is a complete understanding of the pigmentation in syphilis and in retinal hypertension. Retinitis pigmentosa, retinitis punctata albescens and choroideremia should be separated and not grouped as evidence of a single cause until the cause has been convincingly demonstrated and proved.

DISCUSSION

DR. W. E. FRY, Philadelphia: There has been some difference of opinion concerning the origin of the pigment in the fundus. Some have considered that the pigment originates in the pigment epithelial cells and migrates from there into the choroid. Others have considered that the pigment originates separately in the retina and in the choroid. The latter opinion is probably correct. By means of the dopa reaction Black found that only epidermal cells gave a positive reaction; the mesodermal cells gave a negative reaction. He therefore concluded that the pigment in the choroid results from migration from the retina. However, embryologically it has been shown that the pigment appears very early in the retina, i. e., at the fifth week, and very late in the choroid, at the fifth to the seventh month after the development of the lamina vitrea, and that it appears first in the outer layers of the choroid. In the pigmented epithelium the pigment arises on the cytoplasm and is in the form of a melanoprotein. Pathologically, pigment may appear in other cells, as in fibrocytes and also in clasmatocytes of the retino-endothelial system. The pigmentation of the former cells is always finer and less dense than that of the latter. In certain conditions, such as disturbances of nutrition, the chromatophores, which normally have branched processes, lose their processes and have the appearance of clump cells. This has led various authors to believe that these two cells are variations of the same cell. The pigment in the eye is not an inert substance but has certain irritative qualities, and immunologically it is capable of acting as an antigen. The former ability is shown when pigment is injected intra-ocularly, when it produces a certain amount of retinal degeneration. The latter quality has been seen in relation to sympathetic ophthalmia.

Certain analogies have been drawn between dermatologic and ophthalmic conditions. The retinal pigment epithelium has been compared to the epidermis, and the uveal tract to the corium. A particularly important relationship has been shown between angioid streaks of the retina and pseudoxanthoma elasticum. Each of these conditions is rare in its field, and their occurrence together shows a relationship of great importance. There have been certain facts brought out concerning pigmentation in the skin and vitamin C and copper. There are cutaneous conditions, namely, scurvy and Addison's disease, that have as one

feature increased pigmentation. It has been found that the administration of vitamin C will prevent the hyperpigmentation shown in these diseases. An idea of the site of the reaction is obtained by the use of dopa. Vitamin C interferes with this reaction. In the formation of pigment, copper apparently acts as a catalyst, and it has been shown that a definite proportion exists between the amounts of pigment, vitamin C and copper present in the skin. In view of the fact that when pigment is introduced into the eye it causes retinal degeneration, vitamin C therapy may be of use in the treatment of conditions characterized by retinal hyperpigmentation, in spite of the fact that the pigment may be only a secondary factor.

DEFECTIVE CENTRAL VISION AFTER A SUCCESSFUL OPERATION FOR DETACHMENT OF THE RETINA. DR. ALGERNON B. REESE, New York.

It is common experience that after a successful operation for detachment of the retina the central vision is frequently found to be defective. The statistics of Dunnington and Macnie, compiled from cases of retinal detachment in which operation was performed, observed at the Institute of Ophthalmology, New York, were reviewed with the idea of determining what percentage of the patients in whom reattachment occurred regained normal central vision and whether or not the duration of the detachment affected the central vision after reattachment. Of the 55 patients in whom reattachment occurred, 18, or 32 per cent, obtained central vision of 20/30 or better. There were 32 patients in whose cases the duration of the detachment had been one month or less, and of these, 12, or 38 per cent, obtained central vision of 20/30 or better. There were 23 patients in whose cases the duration of the detachment had been over one month, and of these, 6, or 26 per cent, obtained central vision of 20/30 or better.

From these figures one can deduce that approximately normal vision (20/30) is obtained in only about one third of the cases in which operation for detachment of the retina is successful and that the duration of the detachment is a factor in the visual result.

From microscopic examination in the routine cases of retinal detachment in which the eye is enucleated, it is well known that cystic spaces occur in the macular region and, to a less extent, elsewhere in the retina. This change is usually considered a late sequela, and its occurrence has been noted almost invariably in eyes in which there was some other pathologic change, so that it is difficult to say to what extent detachment alone was responsible for the cysts and to what extent other factors were the cause.

Twelve patients with simple serous detachment of the retina ranging in duration from less than two weeks to four years all showed cysts in the macula. It is here that the cysts seem to appear first and to attain their largest size. In the cases in which the detachment is of longer standing—of three months' duration or longer—they are frequently also found elsewhere in the retina. They are situated primarily in the external plexiform layer, but they are often seen to a lesser degree in the internal nuclear layer. At first the retinal elements seem to be separated merely by edema, which localizes into small cystic spaces.

These coalesce so that ultimately the cysts become larger and separated from contiguous cysts by thin septums of glial tissue. The content of the cysts is usually a homogeneous, colorless fluid which stains weakly with eosin in some instances, particularly when the condition is of long standing. This, no doubt, represents an increase in the albuminous content due to inspissation.

Cysts in the macula or elsewhere in the retina cannot be recognized clinically as such by the ordinary ophthalmoscopic examination. They may appear as poorly demarcated foci ranging in color from grayish to yellowish white or as a rarefied area described usually as a "hole." Vogt has recognized them as cystic spaces by means of red-free light and has described their appearance in cases of retinitis pigmentosa, thrombosis of the central vein and detachment of the retina.

In most instances, what is diagnosed clinically as a hole in the macula represents conglomerate or confluent cysts.[1] This statement is based on the following facts:

1. One often sees in the center of a so-called hole in the macula a foveal reflex, which apparently comes from the anterior limiting layer of the cyst.

2. By means of red-free light, or by spectral illumination with the ophthalmoscope, one can usually see cystic spaces surrounding the so-called hole, and sometimes it can be detected that the irregular borders of the hole are continuous with the surrounding cysts. Occasionally, septums can be seen in the hole.

3. Actual holes in the macula almost never are seen in microscopic sections, whereas cystic spaces are common.

The following conclusions may be stated:

1. Cystic degeneration of the macula accounts for the poor central vision after the reattachment of a detached retina which involved the macular region.

2. The defective central vision frequently found after a successful operation for detachment of the retina suggests that macular detachment with formation of cysts was present, even though it might not have been recognized clinically.

Cysts or holes may also appear in the macula as follows: 1. Following trauma. In this condition the cysts are thought by most observers to be due to localized edema, which leads to pressure atrophy of the affected retinal element, with the formation of small cystic spaces, the walls of which form larger ones by confluence. Sometimes the cystic spaces are filled with hemorrhage. A hole may be present after commotio retinae. 2. As a complication of iridocyclitis. In this condition toxins apparently reach the macular region and give rise to inflammatory edema. These cysts may be transitory and may reappear. 3. Accompanying retinitis pigmentosa. In this condition there may be cystic formations in the macula, usually in the early stage of the disease. These may be transitory and are usually attributed to edema. 4. Following occlusion of the central vein of the retina. Cysts in the macula are extremely common in this disease. They are due to edema, and sometimes they are filled with blood. 5. As a complication of retinal arteriosclerosis. This condition occurs in older persons, and the cysts are attributed to edema due to arteriosclerosis. They may appear in the early stage of senile macular degeneration. Arteriosclerosis due to syphilis may also be the cause of macular cysts.

DISCUSSION

DR. CLIFFORD B. WALKER, Los Angeles: The cystic degeneration that occurs in Henle's layer, that is, the plexiform or internuclear layer, was first noted by Bruno in 1902 in connection with melanosarcoma of the choroid. It was repeatedly noticed until 1910, and in 1916 Vogt noticed the cystic degeneration taking place at the macula in the case of carcinoma, and he wished to connect it with the separation of the retina, with the thought of possibly showing the mechanism of the detachment.

One can see that the layer of Henle, which is marked at the macula and constitutes most of the tissue there, because the two layers have been pushed aside, has degenerated in such a way that there are a top layer of limiting membrane and a bottom layer, and there are intermediate stages, as can well be imagined, which might affect the external limiting membrane in such a way that three layers may occur—laminated holes, I shall say, for lack of a better phrase now.

I shall speak concerning the pebbling that is noticed around the hole. If this is cystic in character, it is exceedingly regular in outline. But the cysts, as is noticed, are not so regular, so uniform or so able to indicate the pebbling like Scotch grain leather.

The pebbling is due to toxic effects. When one watches the same process in capillary degeneration, there is edema, with early stages of cystoid degeneration. But in regard to the pebbling that occurs under the conditions found in the retina, it seems to me reasonable to regard it largely as a pattern of the basework of the retina and as an underlying edematous and pyoid degeneration rather than as cysts in that particular spot and with that particular arrangement.

DR. HARRY S. GRADLE, Chicago: The lack of central visual acuity after a successful operation for detachment of the retina may be due to one of three things. In the first place, there may have been a preoperative disease involving the macula. This possibly might have been seen beforehand, but if the detachment was sufficiently extensive to elevate the macula it would have been impossible to observe it.

Second, a postoperative change in the macula is usually subsequent to hemorrhage. Hemorrhage into the macula following operation for detachment of the retina is not uncommon.

Third, patients who on ordinary ophthalmoscopic examination show a normal macula may have a lack of central visual acuity and a definite central scotoma often from 3 to 5 degrees in size.

Since April I have observed 4 cases of defective central vision following a successful operation for detachment of the retina in which I have been able to detect the cause of the failure of macular vision. I have not been able to see these cysts with the ordinary ophthalmoscope but have seen them with the Friedenwald slit ophthalmoscope when used, as Dr. Reese has described, with retro-illumination (if one wishes to call it that) and particularly when used with the red filter—and I said red, not red-free. The details of such cysts do not appear with the red-free filter but appear clearly when the red filter is used.

In these 4 cases the cysts lay directly in the macula and varied from one quarter to a little more than one third of a disk diameter in size. In every instance they were regular and oval, vertically oval. This was

probably a coincidence. At no time did I find the irregular walls as in the last picture shown by Dr. Reese. The cysts were elevated, and the elevation could be detected by the use of the extremely fine slit. There seemed to be a thin but definite anterior wall, which, apparently, varied in thickness in the different cases. The surrounding macula was flat, and, as far as could be detected, there was no elevation of the retina in or around the macula except at the cyst.

In this connection, may I speak of one other observation that I have made in 2 cases? On the fifth to the ninth day subsequent to detachment of the retina or to operation for detachment of the retina there has appeared massive detachment of the choroid. This persisted in 1 instance for four weeks. In another instance it persisted for three weeks, and was followed by successful reattachment of the entire retina, with good central visual acuity.

From the mechanical standpoint, it is easy to see why choroidal detachment is a desirable feature, because, whatever surgical procedure one performs for detachment of the retina is for the purpose of producing adhesion between the retina and the choroid, for the production of which adhesion the retina must either be in apposition with the choroid or close thereunder.

If a large amount of subretinal fluid is present, it is obviously impossible for the retina to be caught in the adhesive process and retained there. Consequently, there is the possibility that certain types of retinal detachment may be handled by the artificial production of detachment of the choroid, which, of course, is spontaneously limited.

A New Operation for Chronic Glaucoma. Dr. Otto Barkan, San Francisco.

This article was published in full in the November 1936 issue of the *American Journal of Ophthalmology,* page 951.

Visual Sequela Resulting from Meningococcic Meningitis. Dr. Parker Heath, Detroit.

This article will be published in full later.

Nodular Dystrophy of the Cornea. Dr. Benjamin Rones, Washington, D. C.

Groenouw (*Arch. f. Augenh.* 21: 281, 1890) first described nodular dystrophy of the cornea (*knötchenformige Hornhauttrübungen*) as consisting of numerous small, roundish, gray, nonconfluent opacities in an otherwise clear cornea. These tend to involve the central portions, leaving the periphery clear. The larger nodules cause the epithelium to bulge forward and become irregular. These opacities remain unchanged for many years, usually without inflammatory changes. Both eyes are always affected, and the disease sometimes occurs in several members of the same family.

The type of nodular dystrophy described by Salzmann (*Ztschr. f. Augenh.* 57: 92, 1925) differs from the aforementioned condition in that the nodules are larger and less numerous and are markedly elevated above the surface of the cornea. It occurs usually in women and is not

familial. In most of the reported cases the condition was unilateral, and there was a history of repeated attacks of eczematous keratoconjunctivitis. Salzmann expressed the opinion that this variety of dystrophy is not as rare as the paucity of reported cases leads one to believe.

The lattice type of corneal dystrophy was first reported by Haab (*Ztschr. f. Augenh.* **2:** 235, 1899), who described it as "alphabet keratitis," beginning at puberty and always being bilateral. In this condition the opacity consists of many fine lines arranged in a lattice-like pattern. Dimmer (*Ztschr. f. Augenh.* **2:** 353, 1899) stated the belief that recurrent inflammation is present early in the condition, but Parsons (The Pathology of the Eye, New York, G. P. Putnam's Sons, 1904) did not concur in this opinion.

Numerous theories have been offered to explain the origin of corneal dystrophy. In reviewing the literature one is struck by the number of cases occurring in members of the same family. The most noteworthy example of familial occurrence is Frykholm's (*Klin. Monatsbl. f. Augenh.* **94:** 76, 1935) series, in which 18 of 42 members of a family were affected, over 6 generations. Judd (*Am. J. Ophth.* **16:** 310, 1933) reported 9 cases in 3 generations, and M. Freiberger (Corneal Dystrophy in Three Generations, ARCH. OPHTH. **16:** 257 [Aug.] 1936) reported 12 cases in 9 generations. On the basis of his series, Freiberger called the condition "familial corneal dystrophy." This term should not be adopted, for in a large number of the reported cases the condition was present in only 1 member of a family, with no history suggesting that other members were involved. The authors of the three series just cited consider that the condition is inherited as a dominant trait and is not sex-linked. It occurred with equal frequency among the male and female members of the families and was transmitted through both sexes.

Fischer (*Arch. f. Augenh.* **98:** 41, 1928; **100-101:** 480, 1929) has demonstrated a close relationship between the transparency of the cornea and its water content. If the cornea loses or takes up more than about 20 to 30 per cent of its water content it becomes semitranslucent. This author has also shown that the imbibition of water is related to the integrity of the epithelium and endothelium and that if either layer is injured the cornea becomes permeable to water in both directions. He has observed that the intact cornea maintains an irreciprocal permeability toward water and sodium chloride and that the unidirectional flow is toward the aqueous. However, Krause (The Biochemistry of the Eye, Baltimore, Johns Hopkins Press, 1934) expressed the belief that the epithelial permeability is evidently influenced through the action of the trigeminal nerve. Tagawa (*Arch. f. Augenh.* **102:** 231, 1929) has found that the water content of the epithelium was regulated by the nerves and that after irritation of nerves the amount of water in the cells was increased. Also, after sectioning of the trigeminal nerve the epithelium swelled, and islands of cells were desquamated.

On the basis of this work one can offer the hypothesis that the initial lesion in corneal dystrophy is an irritative lesion in the corneal nerves. This results in increased permeability of the epithelium, with an increase in the water content of the epithelial cells and superficial lamellae and a change from irreciprocal to reciprocal corneal permeability. This

brings about a disturbance in the oxygen metabolism, resulting in necrosis and hyalinization of those cells which are farthest removed from the limbus and aqueous, that is, the cells in the superficial layers of the center of the cornea, as is seen in this type of corneal dystrophy.

In summary, the following points are noted:

1. A review of the literature of the nodular and the lattice type of corneal dystrophy leads to the belief that they are merely variations of the same condition.

2. Although the variety of the pathologic changes is confusing, the frequency with which hyaline masses are seen in the epithelium and superficial lamellae would permit this nodular formation to be called the characteristic lesion.

3. The theories offered in explanation of corneal dystrophy are discussed and found to be unsatisfactory.

4. An explanation of the condition is offered, based on the disturbed permeability of the cornea resulting from an irritative lesion of the corneal nerves.

DISCUSSION

Dr. Mark J. Schoenberg, New York: Very stimulating and interesting is Dr. Rones' attempt to formulate a hypothesis as to the manner in which the nodular lesions of the cornea are formed. Fischer, Tagawa and Krause have contended that the imbibition of water by the corneal tissue is related to the integrity of the epithelial and endothelial layers, that the permeability of the cornea is influenced by the action of the trigeminal nerve, that the water content of the epithelium is regulated by the nerves and that after irritation of nerves the amount of water in the cells is increased. On the basis of the findings of Fischer, Tagawa and Krause, Dr. Rones has advanced the hypothesis that the initial lesion in corneal dystrophy is an irritative lesion in the corneal nerves, which irritation results in increased permeability of the epithelium, with an increase of the water content of the epithelial cells and superficial lamellae. This is followed by a disturbance of the oxygen metabolism, with the formation of nodules and necrosis and hyalinization.

Secondary Cataract, with Particular Reference to Transparent Globular Bodies. Dr. Alfred Cowan and Dr. Wilfred E. Fry, Philadelphia.

This article was published in full in the July issue of the Archives, page 12.

Use of Concentrated Preparations of Epinephrine in Glaucoma, Iritis and Related Conditions. Dr. Meyer Wiener and Dr. Bennett Y. Alvis, St. Louis.

The use of strong preparations of epinephrine over a period of years in 13 cases was recorded. This experience and the rich literature on the subject make possible certain conclusions which constitute a fairly comprehensive set of working rules for the use of this important therapeutic agent.

1. A concentrated solution of epinephrine hydrochloride is a powerful mydriatic capable of lowering the ocular tension, producing an acute rise of the general tension and causing severe pain and, occasionally, general symptoms; hence, when it is first used conditions should be such that the patient can be observed for one or more hours.

2. Epinephrine is a valuable aid in cases of chronic simple glaucoma in which miotics must be used for prolonged periods in lieu of the preferred surgical measures.

3. Chronic secondary glaucoma generally does not yield to epinephrine, but may do so, and a trial is justified.

4. After operation for glaucoma, epinephrine may be effective in bringing about a state in which the tension remains normal for long periods without treatment.

5. Glaucoma, after needling of cataract or the membrane of an after-cataract, has been controlled in a large percentage of cases by epinephrine, given with or without miotics.

6. Epinephrine is invaluable in breaking up adhesions in cases of iritis and uveitis.

7. The most convenient form for office and prescription use is an ointment in a water-soluble base (tragacanth jelly) or in an oily base (petrolatum and hydrous wool fat).

DISCUSSION

DR. E. C. ELLETT, Memphis, Tenn.: Ophthalmologists have gone a long way in the knowledge and application of epinephrine since Darier first introduced it in the field of ophthalmology in 1896 as an agent to dilate the pupil and reduce the intra-ocular tension. The present manner of employing it may be said to date from 1922, when Carl Hamburger advocated the use of epinephrine in the treatment of simple glaucoma, later developing the preparation which he called glaucosan (dextro-glaucosan). This is a combination of dextrorotatory epinephrine and methylamino-acetocatechol. This was intended for subconjunctival injection.

Continuing his researches, he developed a mixture of levorotatory epinephrine and methylamino-acetocatechol, which he called levoglaucosan. This proved to be active enough to use by instillation only. It causes dilatation of the pupil and a decrease in the intra-ocular tension.

The method of instilling levoglaucosan is simple. The eye is anesthetized with phenacaine hydrochloride or butyn, and then, with the patient in a recumbent position, 2 drops of the solution as furnished in the ampules is instilled into the cul-de-sac. The patient is directed to rotate his globe in the four cardinal directions while the lids are held open, which allows the drug to come into free contact with the globe. Drops are instilled again from three to five times at intervals of fifteen minutes. In a few moments after the first drop is instilled the sclera should become porcelain white, the aperture of the lid widen and the pupil dilate regularly, while a zone of ischemia usually develops on the lids, cheek and temple. Practically always there is a distinct drop in the tension. Sometimes the cornea appears as though glistening powder had been sprinkled on it. This appearance soon passes off.

There has recently been available, due to the efforts of Dr. John Green, a synthetic epinephrine bitartrate, dispensed as a powder in glass ampules. Each ampule contains 0.091 Gm. of the drug, and when dissolved in 2.5 cc. of water this makes a 2 per cent solution. At the suggestion of Dr. Melville Black, my associates and I have used this in the powdered form, taking up a small amount on a very small eye curet. The solution is not stable, but the powder is, and the use of the powder is satisfactory and economical.

As regards these preparations of epinephrine, my associates and I can speak favorably of their use in iritis, especially in those troublesome cases in which the tension is increased. The pupil can be widely dilated, adhesions broken and the tension usually reduced, at least, never elevated. The dilatation can often, but not always, be maintained with atropine. Subsequent applications of epinephrine seem much less effective than the first one. I have published my observations, with reports of many illustrative cases, to which I could add many more. The effect in breaking adhesions and fully dilating a rebellious pupil is striking, and the pain, when present, is usually relieved.

Without apparent cause the application is sometimes painful. My associates and I administer the drug with the patient lying down, and first give 10 grains (0.65 Gm.) of acetylsalicylic acid. In addition, some opiate should be at hand for hypodermic injection. The unpleasant effect does not interfere with the dilatation of the pupil, and it is not due to hypertension.

In glaucoma the results of the use of epinephrine are not so good. Sometimes, but not always, the tension in simple glaucoma can be reduced by this drug and can subsequently be kept under better control with miotics, but the result is not constant; at best, epinephrine is only an adjuvant to miotics, and sometimes it will precipitate a severe attack of acute glaucoma. The form which was introduced as especially effective in acute glaucoma, namely, a 10 per cent solution of histamine, has proved to be a flat failure, in my experience. It neither contracts the pupil nor lowers the tension.

NAEVUS FLAMMEUS AND GLAUCOMA. DR. K. L. STOLL, Cincinnati.

This article will be published in full later.

SCLEROMALACIA: REPORT OF A CASE. DR. FREDERICK A. KIEHLE, Portland, Ore.

Rather recently, van der Hoeve (abstr., *Zentralbl. f. d. ges. Ophth.* **26:** 504, 1932) reported several cases of scleral disease under the title of scleromalacia perforans, which fall under a classification that Rochat (abstr., *Zentralbl. f. d. ges. Ophth.* **29:** 663 and 664, 1933) had termed scleritis necroticans. Van der Hoeve made the suggestion that this condition may be more frequent than is commonly suspected and emphasized its association with the severe polyarthritis present in the same patient.

The case to be briefly described here is one of interest as further emphasizing this connection. It is unusual in that the condition falls into none of the known classifications of scleral diseases and varies in certain particulars from the condition in van der Hoeve's cases, in which per-

foration occurred. No condition resembling it can be discovered in reports in the literature, nor has a case of any similar condition been encountered in the experience of several distinguished confrères who have examined the patient.

Since the case is evidently one of degeneration of the sclera, the term scleromalacia (Professor van der Hoeve's nomenclature) seems most appropriate.

The patient, a woman, is now 64 years old. She has been a victim of rheumatism since the age of 21. She took to her bed nine years ago because of a fractured hip, and has been there continuously since because of deforming polyarthritis. Practically all the joints are affected. Her hands are gnarled, her feet less so. Her spine is nearly rigid. There is some movement of the knees, elbows and shoulders, and she can stand and even take a few steps if aided. Tophi are present in several joints.

In 1929 both eyes showed similar changes, which were more marked in the left eye. The first glance seemed to reveal a huge anterior staphyloma, though a moment's examination showed the fallacy of this observation, as all the bulbar measurements were normal. The unusual appearance was due to the color of the sclera, which had lost its normal whiteness and was slate gray. At a distance the sclera resembled the cornea with the gray iris behind it. The conjunctiva appeared to be intact. The episcleral tissue throughout, after a stage of edematous swelling, has, with a gradually receding margin, been completely absorbed.

When first seen, the cornea and the other media were clear and permitted satisfactory intra-ocular examination. Such examination revealed no abnormality of the fundus. The tension has never been elevated. As the years passed other signs of degeneration occurred—both lenses are now cataractous, and both eyes are sightless except for perception of light. The left cornea has in recent years shown generalized clouding and roughening but without ulceration. I suspect that uveitis has existed, but it has been so insidious as to cause no symptoms. A recent development is the occurrence in each iris of multiple radiating perforations, two or three in line, each perforation measuring from 1 to 3 by 1 mm. These radiating lines are separated by distances of 3 or 4 mm. around each iris. These perforations appear to be ruptures through the thin, atrophic iris.

At no time has pain been an important feature of the condition. Such discomfort as the patient has suffered was described rather as "burning" and "irritation." As to the inception of the ocular symptoms, her recollection is that the trouble in the eyes began with the common sensation of a foreign body in the eye. There is no history of trauma or of previous iridocyclitis. The palpebral conjunctiva appears normal, as do the lids and other adnexa. Bulbar movements are normal. Thorough general physical examination by a competent internist revealed no systemic disease apart from the chronic arthritis. Questions as to the patient's customary diet suggested no vitamin deficiency.

It is significant that all conditions of this type involving degeneration of the sclera have occurred in patients suffering from arthritis deformans. It is suggested that defective metabolism in the fibrous tissue and insufficient elimination of waste are a possible basis of arthritis deformans. This may be a factor in scleromalacia. Friedenwald

(*Am. J. Ophth.* **4:** 433, 1921) stated that arthritis deformans is regarded as a chronic infection. He, with a number of other writers, quoted Osler (The Principles and Practice of Medicine, ed. 11, New York, D. Appleton & Company, 1920, p. 1160): "Arthritis deformans is regarded as a chronic infection caused by certain varieties of streptococci and secondary to a focus of infection somewhere."

DISCUSSION

DR. WILLIAM L. BENEDICT, Rochester, Minn.: I saw the patient in April of this year with the condition as depicted on the screen. It seemed to me that not only was the sclera undergoing a gradual process of absorption but that in certain areas there was an infiltration which could not be looked on as inflammatory but was probably a condition that one frequently sees in cases of malacia, in which there is a deposit of fat and other abnormal tissues in the region.

Furthermore, through the very thin sclera and through the nearly transparent conjunctiva appeared the atrophic uveal tract. The slate color of the eyeball evidently was due to the pigment in the uveal tissue showing through. The mottled appearance was due to the infiltration of the region which once had been occupied by the sclera.

It was striking to note the marked changes in the sclera associated with such an insignificant change in the cornea. For many years after the scleral degeneration had been progressive the cornea had remained nearly clear, even in its marginal portions.

One recognizes well the changes that occur in the choroid in cases of inflammatory diseases of the sclera and in those of scleritis and episcleritis. I consider the change of the choroid, the spaces in the iris and the opacity in the vitreous as secondary to the disease in the choroid and not necessarily a part of the process in which the sclera was the most prominent tissue affected.

The relationship between the disease of the eye and the arthritis or fibrositis is more than incidental. Evidently the inflammatory process had progressed much further in the joints and in the muscles, and evidently some changes in the bones had taken place. I can only agree with Dr. Kiehle that both the changes in the eye and those of the bones must be secondary to some primary cause.

GERMAN OPHTHALMOLOGICAL SOCIETY

Fifty-First Annual Meeting, Heidelberg, July 6-8, 1936

DOCENT DR. M. BÜCKLERS, Tübingen, *Reporter*

TRANSLATION BY PERCY FRIDENBERG, M.D., New York

Opening Session

At the beginning of the session the Dean of the Medical Faculty announced that the University of Heidelberg, in connection with its Five-Hundredth Anniversary Jubilee, had conferred the degree of honorary doctor on Professor Van der Hoeve, of Leyden, Netherlands.

This honor, of which Professor van der Hoeve expressed his appreciation, met with marked and general approval. The presiding officer, Professor Löhlein, then welcomed the visiting colleagues who were present in large numbers from abroad, as well as from other cities of the Reich. He called attention to the necessity and importance of the experience and daily detailed work of the practicing ophthalmologist, as well as the experimental work and laboratory data of the research worker. Many questions remain to be answered, as shown, among other things, by the wealth of data in regard to hereditary diseases of the eye collected by ophthalmologists all over the world to a degree unequaled in any other specialty and so greatly to be envied. Since the prevention of hereditarily diseased offspring and descendants has become a matter of legal regulation and an old requisite of preventive medicine has been fulfilled, it is necessary to take stock and see to what extent ophthalmologists are standing on sure ground and what problems, on the other hand, still await their solution.

The president then extended the thanks of the society to the official reporters, Drs. Oehlkers, von Verschuer, Waardenburg and Clausen, for their cooperation in preparing and presenting the fundamental facts on this subject.

First Scientific Session

Monday, July 6, 1936, 8 a. m.

J. VAN DER HOEVE, Leyden, Netherlands, *Chairman*

SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY: I. HEREDITY FROM THE GENERAL BIOLOGIC STANDPOINT. DR. F. OEHLKERS, FREIBURG IN BREISGAU.

Under certain circumstances the general science ophthalmology is obliged to elaborate material which is not significant or important from the practical standpoint, in order to get and guarantee theoretical progress as quickly as possible. This drawback can be partially overcome or at least neutralized to some extent by cooperation with physicians and breeders. To characterize the present status of research in heredity, three fields have been chosen. The first is concerned with the chromosome theory. The proofs of this theory start with the mendelian law and show, to begin with, that the mechanism of division of the cell nucleus at maturity guarantees the recombination of the rudimentary units postulated by Mendel's law. Then Morgan's theory of linear arrangement of the genes in the chromosome was discussed in line with recent observations and experimental research on anomalies of chromosomes and roentgen ray translocation and was shown to be correct as indicated on chromosome charts. On the other hand, the hypothetic distances between the genes, estimated from the "crossing-over" values, do not correspond to the data of the latest investigations. Accordingly, the chromosome charts are to be considered as purely graphic representations of the crossing-over values. The significance of the chromosome theory for the construction of a theory as to the genetic condition of a population was then considered. If this condition is connected with the parents by way of division of the cell nucleus at maturity and sexual propagation, one must expect basic variation in the hereditary

characteristics of successive generations in case of a heterozygotic foundation. If, on the other hand, descendance is connected with the starting organism merely by way of the usual mitotic division, both links of the chain are equal from the standpoint of genetics. The question of development of the hereditary anlage was then considered in the light of latter day research. Recent experiments with mosses (von Wettstein) seem to show that a dominant and a recessive quality, respectively, depend in many instances on the quantity of genes and that alteration of this factor may bring about a reversal of type. There was a short reference to hereditary variation, the frequency of appearance of new hereditary rudiments and the possibility of increasing the speed of mutation by such agencies as heat and the roentgen rays.

II. Inheritance of Disease from the General Medical Standpoint. Dr. O. von Verschuer, Frankfort-on-Main.

What rôle, if any, is played by a hereditary pathologic tendency in the development of a disease? One must, first, put in a separate division those diseases which are, without exception, of hereditary provenance. The conditions are determined with much greater difficulty in the case of diseases which may occur as hereditary and nonhereditary forms, respectively, with the same or a similar clinical picture.

The differential diagnosis of these forms is of great practical importance, and further working out of the knowledge of this clinical factor is needed. The second question involves the construction of the genotype. Besides the theoretical questions previously considered to be of a special biogenetic bearing, one must consider as of most practical importance a decision as to whether the hereditary predisposition to a given disease is specific or nonspecific. The third question is that of the relative significance of inheritance and environment as etiologic factors in disease. A few examples were adduced to show that the conception of the relation of heredity and environment is to be understood variously for different diseases and for nosologic reasons. The final question, that of heredoprognosis, depends largely on the more important considerations of heredodiagnosis and requires continued and more exact research into the actual hereditary conditions of families.

III. Heredity in Ocular Disease. Dr. P. J. Waardenburg, Arnhem, Netherlands.

Discussing the data derived from research on twins, the reader suggested, on account of their brevity and accuracy, the terms gonosomal and autosomal, respectively, for rudiments (anlagen) connected with X-chromosomes, on the one hand, and those connected with the other chromosomes, on the other. In ophthalmology, serial investigations which are continuous, without gaps and requiring no rejections, are far behind the case reports in number. However, the mode of investigation by study of individual cases is of practical value in corroborating the insight into the nature of the hereditary basis, afforded by other methods, of many autosomal and gonosomal ocular characteristics. As examples of variation in manifestations in enzygotic twins the reader instanced a decidedly frequent discordance of amblyopia with concordant strabismus, reported by numerous authors; concordance with an eight

year interval in severe tuberculosis of the iris (van der Hoeve); concordance, together with discordance, in syphilitic conditions of the eye (Vogt, Glatzel); marked difference in degree of ablatio retinae pellucida (Weve), and concordance of the length of the base of the skull and the sphenoidal angle in cases of steeple skull and craniofacial dysostosis, with variation in other dimensions of the skull (Waardenburg). Examples of conditions with far reaching concordance in enzygotic twins are exophthalmic goiter; unilateral as well as bilateral hydrophthalmos; homolateral glioma, senile glaucoma, Leber's disease (in affected persons as well as in transmitters) and atypical forms of pigmentary degeneration of the retina and/or choroid. The total refraction, the corneal refraction, astigmatism, epicanthus and strabismus were considered more from the standpoint of mass statistics. Recently, a hereditary basis has been found for many senile changes (Vogt). Many valuable facts were brought out in regard to the very slight nonhereditary spread (*Streuung*) of refraction. Disturbances of color sensation are as yet provisionally attributed to multiple gonosomal rudiments (*Allele*) at various points (loci). Megalocornea, bulbar albinism, "extra-ocular" nystagmus, night blindness associated with myopia, pigmentary atrophy of the retina and Leber's disease were discussed. Among the diseases described only once were: gyrate atrophy; a form of microphthalmos; a form of myopia; a hypoplasia of the stroma of the iris associated with secondary glaucoma; a sibship or kinship with macular degeneration; a similar relationship with night blindness, myopia, amblyopia and nystagmus, and probably a certain form of cataract. In line with these are some diseases connected with disease of the skin or nerves. The various forms of a dominant gonosomal anlage, nystagmus and Leber's disease were discussed and analyzed; and in connection with the last-mentioned condition Lossen's rule, which has a practical bearing in giving counsel on the question of marriage, was discussed. There are probably a number of other more or less regularly dominant gonosomal ocular characteristics. With some caution one may include flocculi iridis and some manifestations in the group of disturbances of ocular motility, of cataracts and of pigmentary degeneration of the retina and choroid, as well as some rather rare variations (polycythemia; atrophy of the optic nerve associated with diplegia and feeblemindedness, and other conditions).

In discussing the dominant autosomal anlagens, the reader presented a series of "harmless" characteristics, but took exception to the idea that all dominant characteristics represent slight variations. A number of corneal and lenticular opacities, the coloboma group and glaucoma are not such variations. Dominant variations involving the organ of vision, *sensu strictiori,* i. e., the eye and the optic nerve, serious conditions threatening blindness and destruction of the globe, include glioma (exceptionally), detachment of the retina, pigmentary degeneration of the retina, macular degeneration and infantile atrophy of the optic nerve. Serious irregularly dominant diseases include angiomatosis, steeple skull and other anomalies. Most pathologic manifestations in the field of ocular motility and of the lacrimal apparatus are dominant. Definite knowledge of the heredoprocess (*Erbgang*) in recessive autosomal anlages depends largely on a statistical accumulation of data gained from a study of marriages of persons related by blood. Such

was the case with pigmentary atrophy of the retina, albinism, achromatopsia, hydrophthalmos, microphakia, hemeralopia of Oguchi and juvenile amaurotic idiocy. As recessive characteristics were noted a whole series of anomalies which may affect any ocular part except the lacrimal apparatus and the sclera. The most serious are the disorders of the organ of vision, the visual apparatus itself. The reader showed a number of pictures illustrating the spread of such disorders in regions where intermarriage is common (*Inzuchtgebieten*).

In conclusion, considering the difficulties in the way of accurate investigation of heredity in human beings, it would be of the greatest value if in the future more and more attention were paid to possible intermediary stigmas of heterozygotes with recessive characteristics, and if, as far as possible, a search were made for marriages of transmitters of characteristics in order to find out whether there are any differences as to the degree, extent and interval of time between heterozygotes and homozygotes. In the course of the report examples of change of dominance were cited, and some differences between the white and the yellow race (Japanese) were discussed.

PRELIMINARY ANNOUNCEMENTS AND DISCUSSION OF REPORTS I, II AND III. RESEARCH INVESTIGATION OF THE ORIGIN OF AMETROPIAS. DR. PAUL, Lüneburg.

According to Steiger's "mosaic" theory of heredity, anomalies of refraction of low or medium degree develop on a basis of fortuitous combinations of unsuitable and disparate optical systems and axial lengths. The reader had shown, as far back as 1935 (at a meeting of the Northwest German Ophthalmologic Society), that his studies of family trees did not support this theory. He presented statistical tables and graphs showing the result of investigation of family trees, which had become much more numerous of late, of families in which the corneal radius of each parent was known, the refraction determined and the axial length of the globe calculated. In one division were grouped those families in which both parents were emmetropic. There was a total of 215 families, with 377 examined offspring in the first filial generation. It appeared that in families in which there was a correspondence in the dimensional disposition (anlage) of the eyes of the father and the mother (104), 22 per cent of the progeny manifested myopia, and 9 per cent manifested a high degree of hyperopia. In families in which this dimensional correspondence between the paternal and the maternal eyes was missing (111), 19 per cent of the offspring were myopic, and 11 per cent had a rather high degree of hyperopia. There was no indication of an increased tendency to the appearance of anomalies of refraction in the filial generation of parents with dimensional noncorrespondence of the eyes, although this would have been expected according to the mosaic theory.

In those families (87) in which paternal generations had shown myopia, myopia was also found in the filial generation in 50 per cent. It is accordingly evident that the development of myopia in the children is influenced by the occurrence of that anomaly of refraction in the parents, and one must accept as a fact the inheritance of a tendency to the development of a certain form of the eye. Analysis of the individual family trees showed, however, that this developmental tendency

was not coupled with what one might call the dimensional heritage of the eye. The tendency to the development of a particular anomaly of refraction may well be attributed to a genetic factor quite different from the hereditary dimensional disposition of the eye. Wibaut has come to precisely the same conclusions from an entirely different order of investigations. In a future communication the reader intends to discuss the significance of the observation that, at least in youth, hyperopia appears to be more frequent in persons with a corneal refraction lower than normal, on the average, and myopia appears to be more frequent in persons with a corneal refraction higher than normal, and to analyze his material according to the principal biologic groups represented.

CORRELATION OF HEREDITARY DISEASE OF THE EYE AND OF THE CENTRAL NERVOUS SYSTEM. DR. BEST, Dresden.

As the eye may be considered as a portion of the brain protruded, one would expect numerous points of relation between the hereditary diseases of these two organs. However, their number shrinks markedly if one applies the conception of correlation in its stricter sense. An ocular disease may be secondary, dependent on a disease of the brain. Again, one may be dealing with localizations of one and the same disease in the eye and the brain, so that one infers the presence not of genotypically different diseases but of phenotypically differing manifestations of the same disease genus. As an example of dependent, secondary disease of the eye one might mention atrophy of the optic nerve in association with steeple skull, acrocephaly or Crouzon's disease. In cases of such association the atrophy of the optic nerve is the result of a previous papilledema. If, on the other hand, one attributes atrophy of the optic nerve to mechanical pressure on the nerve in its passage through the bony canal, due to an unusually low level of the middle fossa, one is dealing, more than ever, with a secondary symptom and not with a correlation. As examples many other analogous syndromes might be mentioned, such as oculomotor palsies in association with basal aneurysms with a congenital anlage, and atrophy of the optic nerve due to pressure of a dilated third ventricle in association with congenital hydrocephalus. Again, one cannot very well speak of a correlation in the genetic sense when disturbances of ocular function are associated with a neuropathy on account of the character of the cerebral localization, e. g., sympathetic pupillary paralysis or paralysis of the abducens nerve in status dysraphicus.

Finally, even when the same hereditary disease befalls the eye and the brain one is not dealing with a true correlation, *sensu strictiori*. This category takes in, primarily, neurofibromatosis, tuberous sclerosis and von Hippel-Landau's disease, as well as Hand-Christian-Schüller's disease, in which the same xanthomatous proliferations are found in the bones of the skull, the orbit, the hypophysis and even the retina. Aside from the aforementioned neoplastic formations, congenital malformations or developmental anomalies of an almost identical character occur in the eye and the brain, as in the clinical picture described by Schiötz as the vascular encephalotrigeminal syndrome. This consists in tortuosity of the retinal vessels or telangiectases of the trigeminal area associated with capillary angiomas in the brain. An example of pseudocorrelation in the motor field is chronic progressive ophthalmoplegia as a heredo-

degenerative disease. According to Marburg, this is probably an analog of progressive spinal muscular atrophy and of progressive bulbar paralysis and presents a unitary disease of the motor system (amyotrophia muscularis progressiva). To sum up these observations, in many instances of associated lesions of the eye and the brain there is no true correlation in the genetic sense. Considering true correlation of hereditary disease of the eye and the brain, this may be noted in one and the same patient or in different members of the same family. The latter form of correlation, for example, the occurrence in a subject of tapetoretinal degeneration while sisters and/or brothers are feebleminded, is perhaps at least as frequent as is correlation in one and the same person. It is not feasible to discuss all correlations which can occur, but one might mention briefly those cases in which correlation does not occur. In this respect, glioma of the retina is remarkable. Children with glioma of the retina—which is undoubtedly hereditary, once it has developed on a basis of mutation—are, as a rule, in all other respects uniformly normal physically as well as mentally in spite of the fact that there is a marked increase in the persistence force (*Durchschlagskraft*) in comparison with the immediate forbear, as indicated by the predominant bilaterality of this disease in the second generation as against a marked preponderance of unilaterality in the first. Conversely, glioma of the brain is rarely, if ever, associated with hereditary disease. Of diseases with correlated conditions may be noted especially feeblemindedness and idiocy, not so much on account of the various associated ocular developmental defects, e. g., errors of refraction, anomalies of structure of the nerve head, a persistent pupillary membrane and concomitant strabismus, but rather because they enable one to posit some basic principles. To begin with, it is worth mentioning that there is no definitely positive correlation on which one can count to any extent. As far back as the end of the last century the older physicians were forced to note how slight a value attached to what they then called stigmas of degeneration, if one wished to draw conclusions from them in reverse as to the condition of the brain.

It may be worth mentioning that in cases of feeblemindedness there is a tendency toward anomalies of the disk, tapetoretinal degeneration and a high degree of hypermetropia, i. e., anomalies in the form and structure of the globe, while degenerative-motor diseases, such as diplegia, are more frequently associated with concomitant squint. One thing is certain: The more marked the mental degeneration, the more frequent and intensive the correlated developmental anomalies of the eye. This appears especially definitely when one instances the true malformations of the brain, such as microcephaly, microgyria and macrogyria, and anencephaly. In these conditions the associated abiogenesis of the eye takes such marked forms as coloboma and analogous defects, aniridia, microphthalmos and anophthalmos. But here again the question arises whether even in such conditions one is dealing with an actual combination of dissimilar genes, with a phenotypically similar manifestation of one and the same disease genus in the eye and the brain or, finally, with a direct and immediate dependence of the ocular malformation on that of the brain. Similar considerations, by the way, apply to the frequent association of high degrees of hypermetropia and a short corneal radius (Kurtz, Vogt) with feeble-

mindedness and microcephaly, in which cases one may infer that a too small anlage in the brain is mirrored by a similar developmental discrepancy involving the eye.

The more one busies oneself with the subject of correlation, the more one realizes that there is still much to be learned before factual material can be grasped and reconciled with the calculations and the comparatively rigid concepts of genetics. In all probability one must take for granted a rather high degree of lability of genes which have arisen or been altered as a result of mutation.

FAMILIAL MACULAR DEGENERATION. DR. GASTEIGER, Frankfort-on-Main.

A survey of 11 cases of familial macular degeneration noted in one family within three generations, with the family tree, was presented. Its earliest occurrence was at the age of 17, and the first indications consisted in slight irregularities of pigmentation at the posterior pole, which later developed into delicate light gray flecks, resulting finally in a large dirty gray area. The successive stages could be observed in different members of the family. No other disturbances were noted, and, in particular, no mental deterioration was observed, in the family. Vision in the initial stage was $5/10 = 5/5$, and it decreased to $1/50 = 2/50$, when marked ophthalmoscopic changes were noted. The reader then reported a case of congenital oculomotor palsy, showing pictures illustrating an associated ptosis. Motility was completely abolished except in a single case in which the left eye showed a slight abduction. In one member of the family there was, coincidentally, a coloboma of the nerve entrance in one eye; besides, nystagmus and astigmatism were noted. The anomaly was checked up positively through three generations, in one of which all the members of the family, without exception, were affected, according to the statements of those members who were examined. Relatives assured the investigator that the disease had also been present in many earlier generations. As the family was of foreign origin, detailed and intensive investigation met with serious obstacles. A dominant heredity may definitely be accepted.

SIGNIFICANCE OF PERCENTAL CALCULATION OF THE DIMENSIONS OF THE EYEBALL IN GENETIC RESEARCH. DR. PAUL, Lüneburg.

The size of the globe and that of its component parts is influenced by age, sex, race and other factors. In order to test these dimensional values correctly and to judge the relation of different eyes to one another, they must always be considered with reference to a normal or average eyeball. This will be facilitated by a representation of the dimensional values of such an eyeball in percental terms. An emmetropic eye corresponding in form with the usual schematic eye—which may be called the original or eo-eye (*Urauge*)—can be calculated percentally as a pure quantitative variation of the latter. In the case of anomaly in form, the calculation must make use of a similarly anomalous formally schematic eye, the "secondary" eye. The corneal radius of the secondary eye is put down as corresponding with the dimension of the original eye (*Urauge*). The axial length of the secondary eye can be calculated as a percentage of the "original" axis, and this value can be applied as an indication of the activity of the lenticular system—

designated as the lens factor (*Linsenwertigkeit*)—which is coupled with it, as both the original and the secondary eyeball are conceived of as emmetropic. From this value it is possible to determine the accommodative power and the position of the lens system, with the use of the formulas which the reader has worked out and which he reported in a previous communication.

Any emmetropic eyeball shows a dimensional variation when compared with a corresponding, suitable secondary eyeball. In case of ametropia a tertiary eyeball is used as the schematic eye. Its axial length is a percentage of the axial length of the secondary eyeball and, as it is coupled with the error in refraction, can be applied to indicate and measure the latter. Indirectly, by means of the tertiary (and the secondary) eyeball, the corneal radius and the axial length of any eye can be calculated in percental terms of those of the original eye, and the refraction of the lens can be calculated from the lens factor (*Linsenwertigkeit*). The eo-eye, or "original" eye (*Urauge*), is not always suitable as a standard of comparison. However, the comparative percental values found by comparison with an "original" eye can be corrected to comparative values in reference to a more suitable test, or comparison, eyeball, by dividing the figure by the corresponding variations of the chosen standard of comparison. In this way one is enabled to express the dimensional values of the eye under investigation in a form which takes into consideration age, sex, race and other factors of its individuality. It is only by means of such a modification (*Umormung*) that the dimensional data for an eye gain practical significance and value for research in genetic statistics.

Calculations of the corneal radius in grown men (1,002) and women (1,334) from the reader's collection of family trees showed that on the average the corneal radius was 1.7 per cent greater in men than in women. The variation polygon of the corneal radii of men and women, respectively, has, as is well known, a different position and a different form. A combined variation polygon constructed from the figures of the eyes of males and females resembles rather a polygon bounded by a binominal curve, it is true; but, as shown by graphic demonstration and illustrations, it is determined absolutely by the fortuitous combination of the eyes of the two sexes. This indecision as to the exact figures disappears completely if for absolute values one substitutes percental comparative figures calibrated with reference to a suitable standard eyeball.

THE THREE FORMS OF FAMILIAL CORNEAL DEGENERATION AND THEIR HEREDITARY TRANSMISSION. DR. M. BÜCKLERS, Tübingen.

The research, carried out in association with cand. med. Gilch, took in 35 localities and 800 persons. The most frequent form of hereditary corneal dystrophy is characterized by glassy crumbly deposits (*Bröckelchen*) which are sharply defined and surrounded by clear corneal tissue (*Fleischer*). This "crumbly" type of dystrophy begins in the fifth year of life in the form of extremely fine radiating lines and progresses slowly in extent and depth, filling a disciform area between 30 and 40 years of age, which later becomes still more dense and wide. The extreme periphery always remains clear. Two family trees, with 68 members affected altogether, show simple dominance.

The other type of dystrophy, which has also been described as "nodular" by Groenouw, also begins in the first decade but with a delicate, diffuse clouding of the entire corneal surface. Very soon flecks appear, which are superficial as far as the middle of the cornea is concerned but which lie deep in the periphery. In later life and old age the cornea is filled with so many dense opacities of this punctate, flecked form (*Fehr*) that the subject is practically blind. This type is recessive. In the lattice-like (*gitterig*) type of dystrophy, which Haab described, there appears rather early the well known system of characteristic double-contoured lines which bifurcate toward the summit of the cornea. These lines correspond to fine clefts in the anterior layers of the stroma, and this causes a peculiar glassy aspect in reflected (*rückfallend*) light. Scattered among these lines are noted nodular thickenings which at various points raise up the epithelium, as in the flecky, punctate type of dystrophy. The family trees showing instances of authentic and undoubted lattice-shaped corneal dystrophy thus far collected reveal a dominant heritage. According to the reader of the paper, a transition from one of these fundamental corneal dystrophies to the other has never been observed. Accordingly, the inclusive term familial corneal degeneration should be dropped.

A Family with Congenital Macular Degeneration. Dr. Jung, Giessen.

A family in which 22 cases of macular degeneration are reported was studied for the first time by Vossius at Giessen as long ago as 1880. The cause of the macular degeneration was not syphilis, as was formerly assumed, but a heredity which is expressed in a decidedly intensified degree by the inbreeding which was widespread in this family. There are a number of clinical details speaking strongly in favor of this theory as to the etiology. These are: first, the congenital nature of the disease; then, the clinical findings, which are always the same, and the uniform localization of the lesions below the fovea; furthermore, the almost exclusively bilateral occurrence, and, finally, the noteworthy fact that the disease occurs in two separate sibships (*Sippen*), for which, however, a common origin was proved. The reader's investigations of 300 persons, far exceeding in number all those studied in previous researches, resulted in the addition of 8 cases to the 14 already reported in the medical literature. In this connection the cumulative occurrence of other developmental defects of the eye, mainly anomalies of refraction, is striking. For instance, hyperopic astigmatism was found in 68 per cent of the cases of macular degeneration; it was present in 10 per cent of the other cases as well. Another interesting feature is the way in which amblyopia occurs intermittently in cases of macular degeneration. The latter disease does not, however, follow the laws for a simple recessive heritage, as has been generally accepted, according to Rieger, nor can one agree with Behr, who assumes an anlage which up to then had been inherited recessively. A stronger claim to correctness is recognized in Rieger's theory, which accepts a dominant factor that assumes a hypostatic behavior toward another dominant factor of a second gene, so that the latter acquires the significance of an inhibitory factor. The reader is inclined to accept Siemens' hypothesis of an irregular dominance.

(*To be Continued*)

Book Reviews

Studies on the Aetiology and Pathogenesis of Cataract Zonularis: An Academic Treatise. By Gunnar von Bahr. Pp. 236, with 4 plates. Uppsala: Almqvist & Wiksells, 1936.

This work on lamellar cataract is a scholarly addition to the excellent monographs which have recently been appearing in Sweden.

Part I is devoted to a review of the literature. Here one learns that, while zonular cataract was probably first described by Schmidt in 1800, it was Jaeger who with the ophthalmoscope first differentiated it from other congenital types of cataract as a form showing opacities in a deeper layer of lenticular fibers which enclosed a transparent nucleus and were themselves surrounded by a normal cortical layer. The diameter of the zonular cataract varies between 5 and 8 mm., and the opacity, which is composed of closely set, tiny dots, is from 0.5 to 0.7 mm. in thickness. There may be "riders." The outer boundary is always sharply defined; the inner boundary is less distinct. This type of cataract may be partial, and rarely double layers of cataract are seen. It may also be associated with a posterior polar, a nuclear or some other variety of cataract. It is usually found in children, the incidence varying from 0.09 to 1.45 per cent, and females are affected more often than males.

The etiology is obscure. Since the fetal lens has a diameter of about 5.75 mm. at birth, it is obvious that this opacity can form either before or after birth. Genealogical studies have shown that the condition may be inherited. Thus, of 443 members of affected families, there were 236 affected persons, many of whom also had defects of the enamel of the teeth and some of whom had suffered from "fits" during infancy. The association of zonular cataract with defects of the enamel was ascribed by Davidson (in 1865) to zonular deformities in two epithelial organs, the lens and the enamel of the permanent teeth.

A summary of the reports of 14 authors revealed that of 743 patients with lamellar cataract, 51 per cent had suffered from tetany, 52 per cent of 711 had hypoplasia of the enamel, and 46 per cent of 714 had rickets.

In his review of the experiments of other workers von Bahr notes that a rachitogenic diet will not cause lamellar cataract, that cataract due to tetany can occur in a person without manifest tetany, that a decrease in the calcium content of the aqueous may be a possible factor in the production of lamellar cataract and that tetany may cause hypoplasia of the enamel in man. Rickets and other general debilitating diseases probably predispose to defects of the enamel. If lamellar cataract has the same cause as hypoplasia of the enamel it must be formed by the end of twelve or eighteen months after birth.

In part II von Bahr gives a detailed description, with protocols and illustrations, of his experiments with rats. This part of the monograph will be of especial interest to research students, as the author used roentgenograms of the knee joint to determine whether rickets was present, and the galvanic current was used to determine alteration in

the neuromuscular excitability. He also examined the lenses both with the slit lamp and with the ophthalmoscope for the presence of opacities of the lens, and the incisor teeth were observed with a corneal microscope for irregularities in the enamel. Analysis of the blood was made a few times to determine the calcium and the inorganic phosphorus content, and a few lenses were examined histologically.

Part III contains the author's conclusions, and in part IV is a summary of his work, which may be briefly abstracted as follows: When young rats were fed a rachitogenic diet continuously, no cataract was formed, but when there was a change to fasting or to a normal diet, cataract often appeared. Rats made rachitic with Steenbock and Black's ration 2965 showed a more marked and prolonged hyperexcitability in reaction to the galvanic current than nonrachitic rats when the diet was changed to one rich in phosphates. The rachitic rats then often showed manifest signs of tetany. In rachitic rats with tetany, cataract generally developed, at least in all cases in which the anodal opening contraction occurred at less than 0.3 milliamperes, and cataract developed in both the manifest and the latent form of tetany. Hypocalcemia was apparently an essential factor for the production of cataract, and the cataract seemed to be similar to that obtained after parathyroprival tetany in rats, appearing first in the superficial layers of the cortex as fine dots and radial lines (as seen by reflected light). The dots were not directly subcapsular, and the capsular epithelium appeared to be unaltered. The earliest cataractous changes were reversible. Often the formation of opacities progressed even when the tetanic condition had disappeared. The lenticular changes appeared partly as vacuoles and partly as radial lines with fine ground glass-appearing zones of discontinuity which were probably due to shrinkage of lenticular fibers. The impaired cortical layers were covered by fresh lenticular fibers so that a zonular cataract was formed. It could not be proved that tetany in the mother caused zonular cataract in the offspring. In attempting to obtain a decrease in the calcium content of the aqueous, subcapsular dots were sometimes obtained, but it could not be proved that these were due to the decrease in the calcium content. The results of these experiments seem to have some relation to pathologic conditions in man.

This is a most satisfactory monograph, since it not only gives a comprehensive review of the literature (over four hundred references are cited) but also presents the results of careful experimental work which was done to obtain further insight into the factors which may be responsible for the production of zonular cataract.

W. F. DUGGAN.

Bulletin of the Ophthalmological Society of Egypt. Vol. XXIX. Thirty-Third Session, 1936. Pp. 248, with illustrations. Cairo: Imprimerie Misr, 1936.

This bulletin records the proceedings of the Ophthalmological Society of Egypt at its thirty-third session held under the presidency of Dr. M. A. El-Kattan on March 27 and 28, 1936, at the Memorial Ophthalmic Laboratory, Giza. The volume contains 248 pages, 28 devoted to the business of the society and 220 to scientific reports by

members. The reports are well illustrated and contain much information of value to ophthalmologists, particularly those interested in external diseases.

A symposium on the pathology and treatment of trachoma is contributed by R. P. Wilson, M. O. Rifai, I. A. Mohamed, Max Meyerhof, M. A. El-Bakly, M. A. H. Attiah and A. F. El-Tobgy, and F. Masoud. Wilson touches briefly on the changes which take place in the conjunctiva and cornea following trachomatous infection, illustrating each important pathologic change with photomicrographs. He states that "the evidence is growing that the earliest sign of trachoma is the presence of intracellular inclusions in the cells of the conjunctival epithelium, the so-called Prowazek-Halberstaedter bodies." Of distinct interest is his finding that the corneal changes of trachoma occur early and that by the time typical lesions have appeared in the palpebral conjunctiva invasion of the cornea with new vessels has already occurred.

I. A. Mohamed discusses the possibility of trachoma of the lacrimal apparatus and concludes that the question must remain in doubt until the virus has been demonstrated in the lacrimal epithelium or until true trachoma has been reproduced by inoculation of tissue of the lacrimal gland or the lacrimal sac. In a study of 16 inflamed glands, the formation of follicles was noted in 2, but in no instance was there definite evidence that the inflammation was trachomatous. After giving a review of the literature Mohamed states the belief that there is strong evidence for trachoma of the canaliculi. Of 405 lacrimal sacs removed from trachomatous patients studied at the Giza Memorial Ophthalmic Laboratory, 131 showed the formation of follicles. Histopathologic study alone, however, is considered insufficient to elucidate the question of trachomatous infection of the lacrimal sac. Seventeen photomicrographs illustrate this report.

The history of the treatment of trachoma in antiquity, and during the Middle Ages in Arabia is the subject of a report of exceptional interest contributed by Max Meyerhof. It appears that the Greeks and, to an even greater extent, the Arabs possessed considerable knowledge of trachoma. They recognized different forms of the disease and its communicability. The Arabs knew of the connection of trichiasis and pannus with trachoma, and their surgical treatment of the disease was not very different from that practiced today.

The treatment of trachoma is considered completely in the reports by El-Bakly and by Attiah and El-Tobgy. These reports are valuable, since it is only in Egypt that any extensive therapeutic tests of the various antitrachomatous agents have been made. Copper sulfate, chaulmoogra oil and expression operation are considered to be of proved value.

Except for one report on fatty degeneration in a trachomatous cornea, the remainder of the volume is devoted to nontrachomatous conditions. Included among the reports are "Iridectomy Technique Modified to Suit Iridotomy Cases," by M. A. El-Bakly; "Elschnig-Török-Stanculeanu's Intracapsular Extraction," by A. Y. Fahmy; "A Peculiar Case of Polycoria," by M. A. El-Bakly; "A Case of Traumatic Total Depigmentation of the Iris," by S. Kamel; "Ectopia Lentis," by M. A. El-Atawi; "Histopathology of a Case of Capsular Glaucoma,"

by A. Y. Fahmy, and "Studies of a Series of Cases of Sympathetic Ophthalmia," by I. A. Mohamed.

The volume closes with a report of the Committee for Arabic Ophthalmic Terms, in which a large number of ophthalmic words and phrases are translated into Arabic. After revision this work will probably appear in dictionary form.

PHILLIPS THYGESON.

Saggi di oftalmologia. By Prof. Q. Di Marzio and associates, vol. 8, Bologna, 1935 and 1936.

The Ophthalmologic Institute of the University of Bologna, directed by Prof. Quinino Di Marzio, has published the eighth volume of "Saggi di oftalmologia." This book is a collection of reprints of forty original articles written by Dr. Di Marzio and his pupils Caramazza, Puglisi, Duranti, Spinelli, Lugli, Filippi-Gabardi, Biozzi and De Petri.

The researches have a vast scope. The subjects include the comparative anatomy of the eye, the biomicroscopy of the lens and the vitreous, studies on tuberculosis of the different structures of the eyeball, roentgenologic investigation of the optic foramen, the bacteriology of the eye, angiotenometry, ocular syndromes due to lesions of the internal carotid artery and of the sympathetic system, intermittent exophthalmos, and observations on the therapy of various ocular lesions.

These articles show the reader the serious scientific interest of the authors and their thorough knowledge of the subject. The literature is well covered and is discussed in a manner showing the alert supervision of Professor Di Marzio in the work of his associates at the Ophthalmic Institute of the University of Bologna.

G. BONACCOLTO.

Senile Cataract: Methods of Operating. Third revised edition. By Dr. W. A. Fisher. Price, $2.50. Pp. 150, with 181 illustrations. Chicago: H. G. Adair Printing Co., 1937.

In this new edition the chapters by the late Professor Fuchs, I. Barraquer, H. T. Holland, J. W. Wright, A. van Lint and O. B. Nugent have been retained. Fisher describes again the operative technic that he formerly used, which is a modification of Holland's and Smith's methods, and in chapter 9 he describes his new method, which he believes lessens the number of cases of ruptured capsule and loss of vitreous.

This method consists in the use of a suction pump. The cup measures 3 by 4 mm. and after being applied to the capsule of the lens it is slowly raised or moved from side to side until the attachment of the lens to the suspensory ligament is torn; then the suction is released, and the cataract is extracted by the technic of Smith.

Fisher found the Hildreth ultraviolet lamp to be particularly helpful in operations for cataract, as it brings out the cortex and capsule with better distinctness than is gained with the usual sources of illumination.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66, Boulevard Saint-Michel, Paris (6e).
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316 Rotterdam, Netherlands.
Place: Cairo. Time: Dec. 8-14, 1937.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33, Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

Midland Ophthalmological Society

President: Dr. T. E. Ashdown Carr, 34, Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

Ophthalmological Society of Egypt

President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3, Midan Soliman Pasha, Cairo.
Time: March 1938.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 8-10, 1937.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.
Place: Lindley'a 4, Warszawa.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: San Francisco. Time: June 13-17, 1938.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Lee W. Dean, Washington University Medical School, St. Louis.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Salt Lake City. Time: May 24-27, 1937.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501 Seventh St., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.
Place: New Orleans. Time: Nov. 30-Dec. 1-3, 1937.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. W. Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Bldg., Atlanta, Ga.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.
Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York City.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. Rosenberger, 221 Fifth St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514 Sixth St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary-Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Edwin W. Burton, University of Virginia, University.
Secretary-Treasurer: Dr. George G. Hankins, 202 Medical Arts Bldg., Newport News.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Samuel T. Hubbard, 294 State St., Hackensack, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. G. Henry Mundt, 30 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Andrew Timberman, 21 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Claude S. Perry, 40 S. Third St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. A. W. Davidson, City National Bank Bldg., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 720 Medical-Professional Bildg., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Hychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LeGrand H. Hardy, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Philip B. Green, Old National Bank Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto, Canada.
Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 Eye St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.